Dissertation on

AN OBSERVATIONAL STUDY TO EVALUATE THE ROLE OF VISUAL EVOKED POTENTIAL IN INDIRECT TRAUMATIC OPTIC NERVE INJURIES AND ASSESSMENT OF THE VISUAL OUTCOME

Submitted in partial fulfillment of requirements of

M.S. DEGREE

BRANCH –III (OPHTHALMOLOGY)

GOVT. RAJAJI HOSPITAL &

MADURAI MEDICAL COLLEGE

MADURAI

The Tamilnadu Dr.M.G.R. Medical University

CHENNAI, TAMILNADU

MAY, 2018

CERTIFICATE - I

This is to certify that this dissertation entitled "**AN OBSERVATIONAL STUDY TO EVALUATE THE ROLE OF VISUAL EVOKED POTENTIAL IN INDIRECT TRAUMATIC OPTIC NERVE INJURIES AND ASSESSMENT OF THE VISUAL OUTCOME**" is the bonafide original work of Dr.D.Sangeetha , in partial fulfillment of the requirement for M.S.,(Branch III) Ophthalmology examination of the Tamilnadu Dr.M.G.R. Medical university to be held in May 2018.

Prof.Dr.S.V.CHANDRAKUMAR, M.S.,D.O
Head of the Department
Department of Ophthalmology
Madurai Medical College, Madurai

Prof. Dr. D. MARUTHUPANDIAN, M.S., FICS., FAIS.,
The Dean
Madurai Medical College, Madurai

CERTIFICATE - II

This is to certify that this dissertation work titled "**AN OBSERVATIONAL STUDY TO EVALUATE THE ROLE OF VISUAL EVOKED POTENTIAL IN INDIRECT TRAUMATIC OPTIC NERVE INJURIES AND ASSESSMENT OF THE VISUAL OUTCOME "** of the candidate Dr.D.Sangeetha with registration number 221413105 for the award of Master of Surgery Degree in the branch of Ophthalmology. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contained from introduction to conclusion pages and result shows 1% of plagiarism in the dissertation.

Guide and Supervisor sign with seal

DECLARATION

I, **Dr. D. SANGEETHA** hereby solemnly declare that, this dissertation titled "**AN OBSERVATIONAL STUDY TO EVALUATE THE ROLE OF VISUAL EVOKED POTENTIAL IN INDIRECT TRAUMATIC OPTIC NERVE INJURIES AND ASSESSMENT OF THE VISUAL OUTCOME"** was done by me.

I also declare that this bonafide work / a part of this work was not submitted by me / anyone else, for any award, for Degree / Diploma to any other University / Board either in India / abroad. This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Master of Surgery degree Branch -III (Ophthalmology) to be held in May 2018.

Place: Madurai (Dr. D.SANGEETHA)
Date:

ACKNOWLEDGEMENT

I express my sincere thanks and gratitude to **Prof. Dr. D. MARUTHUPANDIAN, M.S.,FICS.,FAIS.,** The Dean, GRH and MMC, Madurai for permitting me to conduct this study. I am extremely grateful to **Prof. Dr. S.V. CHANDRAKUMAR M.S, D.O.,** HOD**,** Professor of Ophthalmology and **Dr. K. KAVITHA M.S, D.N.B.,** Associate Professor of Ophthalmology, GRH, MMC, Madurai, for their constant source of support and encouragement for completing this study. I have great pleasure in thanking my beloved guide **Dr. P.SARAVANA SANKAR M.S,** Senior Assistant Professor and all my Assistant Professors of Ophthalmology department at Madurai Medical College, Madurai, for their constant source of cheer and encouragement throughout the study.

I express my deep sense of gratitude to **Dr. B. SRIDHARAN, MD, DM (NEURO),** Professor and HOD of Neuromedicine, **Dr. MANIVANNAN, MD, DM (NEURO),** Professor of Neuro medicine for evaluation of their patients and their support to this study.

I thank the Secretary and Chairman of the Institution Ethical Committee, GRH Madurai.

I am indebted to all the patients, paramedical staffs for their sincere co-operation for the completion of this study.

I am grateful to my fellow post graduate colleagues for their valuable help throughout this study.

I am extremely thankful to my son Master S.Adidev, S.Ishan, my husband Dr.M.Sivakumar, MS(Ortho)., D(Ortho)., My father Mr. S.Devadoss, My mother Mrs. A.Renganayagi and my sister Mrs.D.Saranya and her daughter P.Niyasha and my family members for their constant support.

I also thankful to my friends especially Dr.Soundarya, Dr.Muralikrishnan, Dr. S.Sangeetha, Mr.Ashokkumar, PT.Chinnamma, Mrs.Usha, Mrs.Ramuthai, Mr.Sivakumar.

Above all I thank the almighty for guiding with his blessings throughout this work.

CERTIFICATE FROM GUIDE

This is to certify that this dissertation work titled "**AN OBSERVATIONAL STUDY TO EVALUATE THE ROLE OF VISUAL EVOKED POTENTIAL IN INDIRECT TRAUMATIC OPTIC NERVE INJURIES AND ASSESSMENT OF THE VISUAL OUTCOME "** of the candidate **Dr.D.Sangeetha** with registration number 221413105 , Post graduate Resident in Department of Ophthalmology, Madurai Medical College , Madurai ,was done for the award of Master of Surgery Degree in the branch of Ophthalmology.

Prof.Dr.S.V.CHANDRAKUMAR M.S.,D.O
Head of the Department
Department of Ophthalmology
Madurai Medical College, Madurai

ABBREVIATIONS

VA-VISUAL ACUITY
RAPD-RELATIVE AFFERENT PUPILLARY DEFECT
NLP-NO LIGHT PERCEPTION
CT-COMPUTERISED TOMOGRAPHY
MRI-MAGNETIC RESONANCE IMAGING
IV-INTRAVENOUS
OD-ONCE A DAY
RED-REDUTION
AMP-AMPLITUTE
WK-WEEK
G-GRAM
KG-KILOGRAM
BW-BODY WEIGHT

INTRODUCTION

Hippocrates noted blows to the eyebrow may cause blindness known as optic neuropathy. Optic neuropathy is a potential complication of head or orbital trauma, results in direct or indirect optic nerve injuries.

Most common form of traumatic optic neuropathy is indirect damage to the optic nerve. Incidence 0.5% to 5% of all closed head trauma which is a consequence of Road traffic accidents or falls.

The incidence of optic nerve injury in severe head injury 13%. The site of injury causing blindness is usually forehead or supraorbital ridge less commonly temporal region.

Direct optic nerve injuries caused by penetrating trauma to the optic nerve, disrupt the anatomic and functional integrity.

Indirect traumatic optic nerve injuries produced by energy absorbed by the nerve, without disruption of normal anatomical structures.

Various manifestations of Indirect Traumatic optic nerve injuries:-

1. Optic nerve Avulsion
2. Optic nerve Transection
3. Optic nerve Sheath Hemorrhage
4. Orbital Hemorrhage
5. Orbital Emphysema.

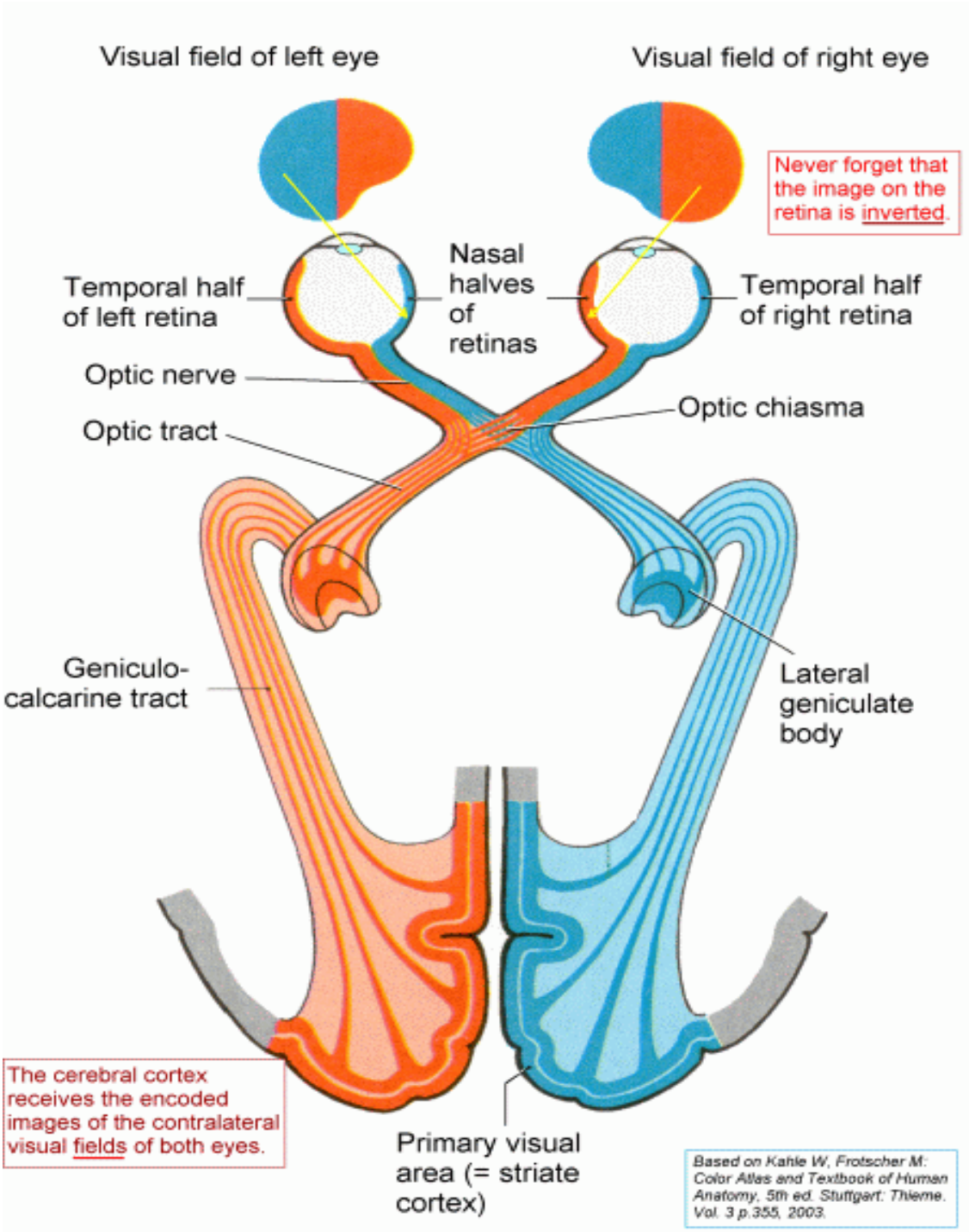
ANATOMY

Visual pathway connects the eyeball and brain, which comprises optic nerve, optic chiasma. Optic tract, lateral geniculate body, optic radiations and visual cortex.

Optic nerve :

Optic nerve known as second cranial nerve starts from optic disc and extends upto optic chiasma. Optic nerve is the backward continuation of the nerve fibre layer of retina, which consists of axons from ganglion cells, afferent fibers of light reflex. Morphologically embryologically comparable to white matter of the brain. Optic nerve does not regenerate when cut because not covered by neurilemma. Optic nerve has very fine millions of fibers 2-10 micrometer in diameter, surrounded by meninges.

ANATOMY OF VISUAL PATHWAY

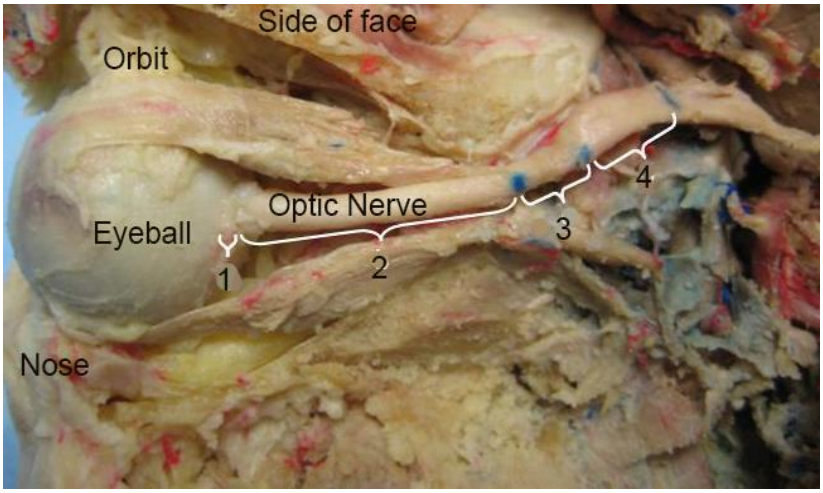


Parts of the optic nerve :

Optic nerve has four parts.

Total length is 47-50 mm

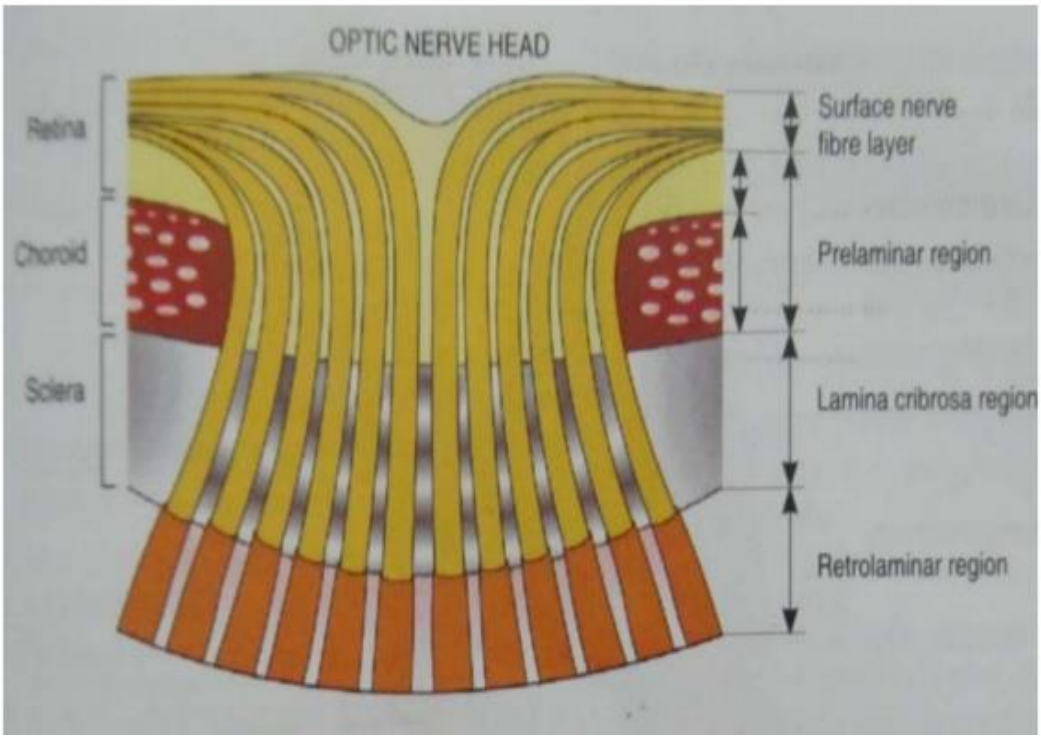
1. Intraocular part (1 mm)
2. Intraorbital part (25-30mm).
3. Intracanalicular (5-9mm).
4. Intracranial (10-16mm)



1. Intraocular part :

This part passes through sclera, choroids and reaches the optic disc. Divides into four portions from anterior to posterior

a) Surface nerve fiber layer
b) Prelaminar region
c) Lamina cribrosa
d) Retrolaminar region



a) Surface nerve fiber layer

Composed of 94% axonal bundles, 4% astrocytes.

Optic disc comprises astrocytes, internal limiting membrane of Elschnig, central meniscus of Kuhut and intermediate tissue of Kuhut.

b) Prelaminar region

Predominant structure of this level are neurons, astroglial tissue, Jacoby separates the nerve from choroids.

c) Lamina cribrosa

A fibrillar sieve like structure made up of fenestrated sheets of scleral connective tissue lined by glial tissue bridges the posterior scleral foramina or scleral canal. Optic nerve fibers leave the eye through these fenestrations.

d) Retrolaminar region :

This portion characterized by decrease in astrocytes and acquisition of myelin supplied by oligodentrocytes. The diameter doubles and passes through sclera.

2. Intraorbital part :

Starts from back of the eyeball and ends in optic foramina. This part slightly sinuous facilitates eye movements. In this region all three layers of meninges and subarachnoid space surrounded the optic nerve. Central retinal artery along with vein crosses the subarachnoid space and enter the nerve on its inferotemporal aspect.

Anteriorly, nerve separated from the extraocular muscles by the orbital fat. Posteriorly near optic foramina, optic nerve surrounded by annulus of Zinn and four recti muscles originates here. The long and short ciliary nerves and arteries surround the nerve before entering the eyeball. Superiorly, ophthalmic artery, superior ophthalmic vein and nasociliary nerves situated. Laterally ciliary ganglion, oculomotor divisions, nasociliary nerve, sympathetic and abducent nerves are situated.

3) Intracanalicular part :

Ophthalmic artery crosses the nerve inferiorly from medial to lateral side in the dural sheath. Sphenoid and posterior ethmoidal sinuses lie medial to it.

4) Intracranial part :

This part lies above cavernous sinus and converges with its fellow to form chiasma, ensheathed by pia mater, Internal carotid artery runs below and medial to it.

Optic chiasma :

Optic chiasma measures 12x8mm horizontally and anteroposteriorly, ensheathed by pia and surrounded by cerebrospinal fluid.

Optic chiasma lies over the diaphragma sellae posteriorly continuous with the optic tracts and forms the anterior wall of third ventricle.

Nerve fibers arising from nasal halves of the two retinae decussate at the chiasma.

Optic tracts :

Optic tracts are cylindrical bundles of nerve fibers running outwards and backwards from posterolateral aspect of optic chiasma. Each optic tract consist of fibers from temporal half of the retina of same eye and nasal half of the opposite eye.

Posteriorly, each optic tract ends in the lateral geniculate body.

Lateral geniculate body :

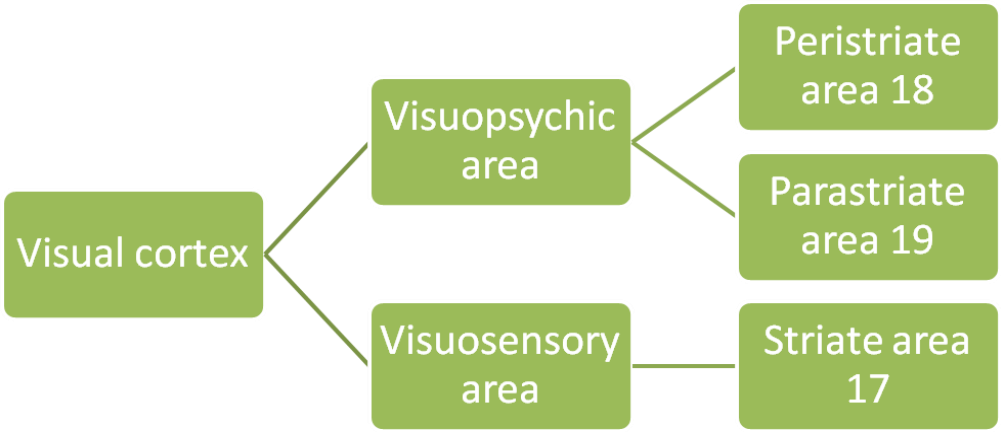
Oval structure situated at termination of optic tracts. Each geniculate body consists of six layers of neurons lies in grey matter alternating with white matter.

Optic Radiations :

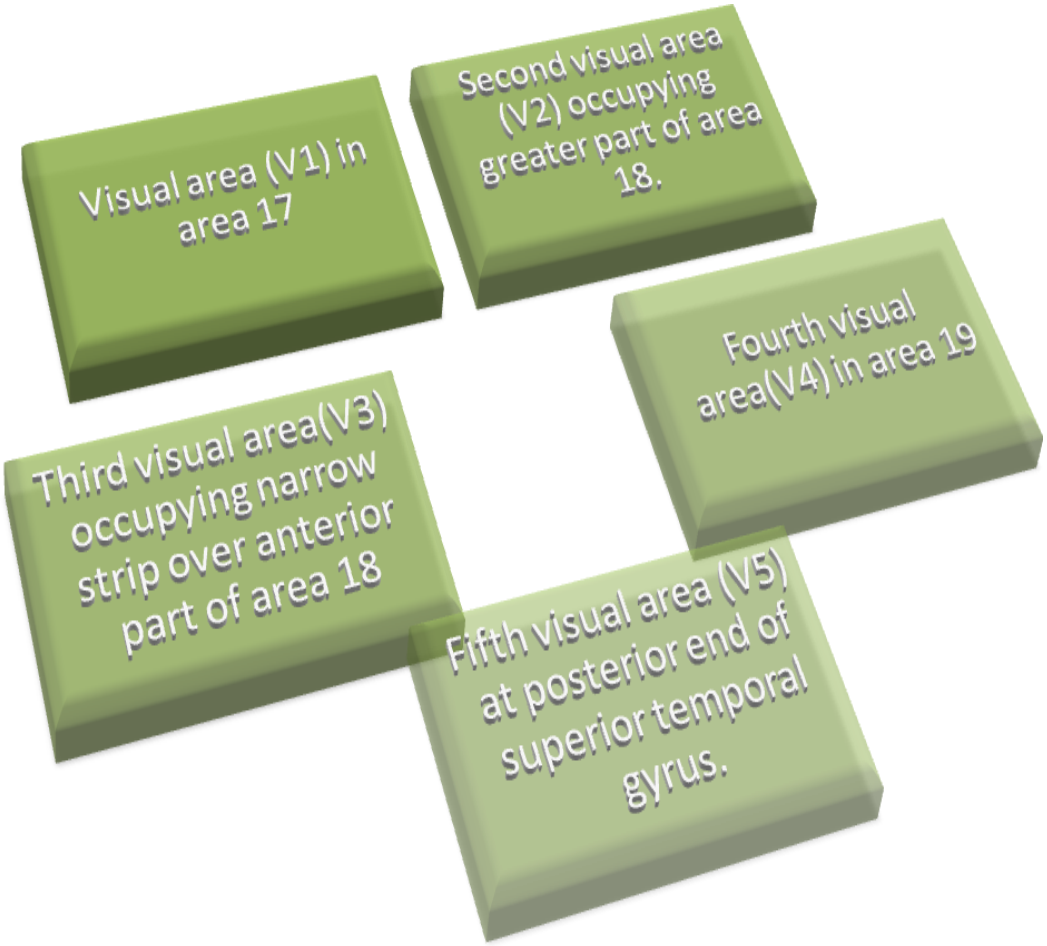
Knowns as geniculocalcarine pathway entered from lateral geniculate body to visual cortex. They pass forwards and laterally through the area of Wernicke as optic peduncles anterior to lateral ventricle and traversing retrolenticular part of internal capsule, behind the sensory fibers and medial to auditory tract.

Visual cortex :

Visual cortex located on the medial aspect of the occipital lobe, near the calcarine fissure may extend on to lateral aspect of the occipital lobe, limited by semilunar sulcus, sulcus lumatus.

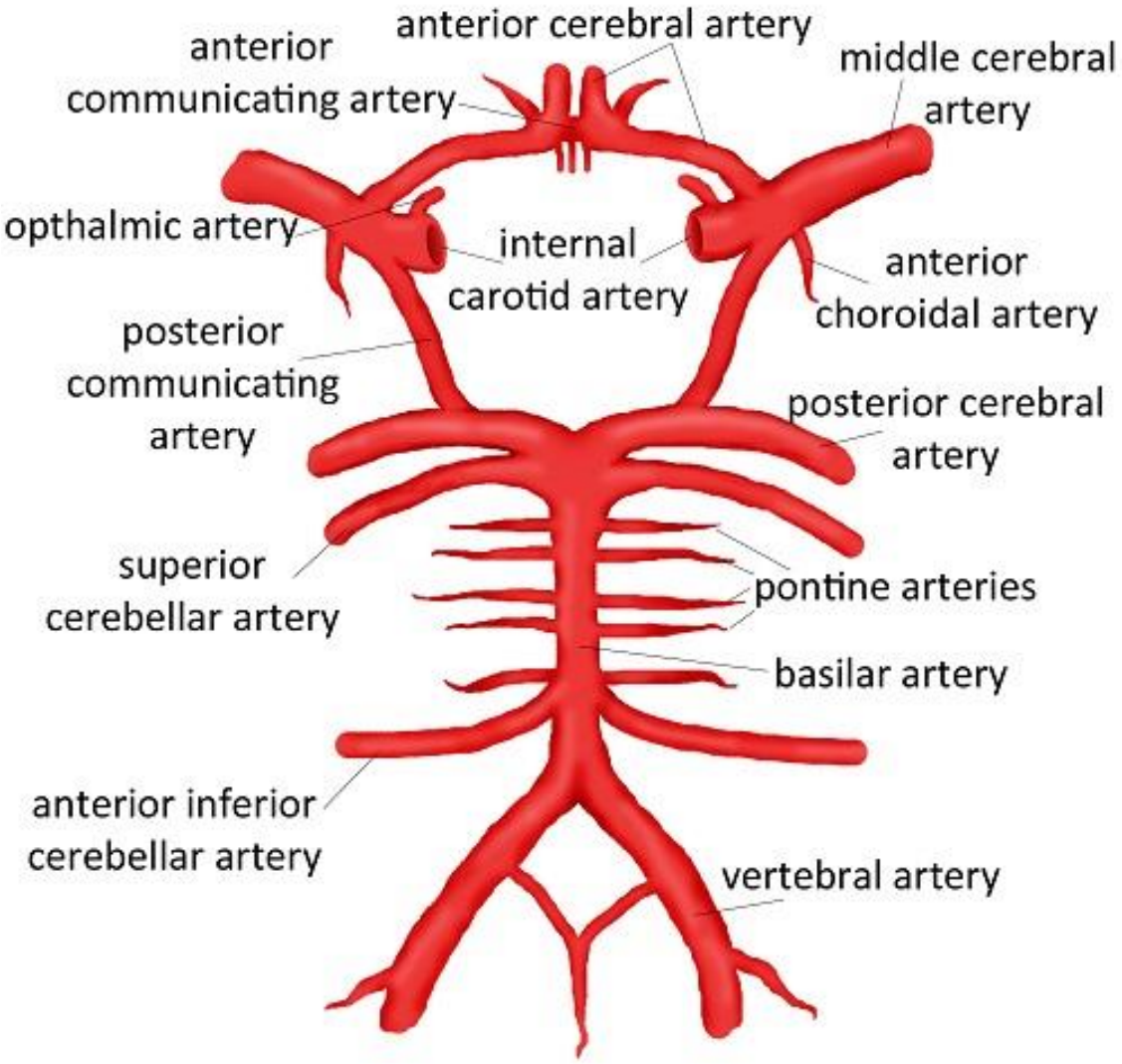


MODIFIED NOMENCLATURE RECOGNIZING FIVE VISUAL AREA



Blood supply of visual pathway :

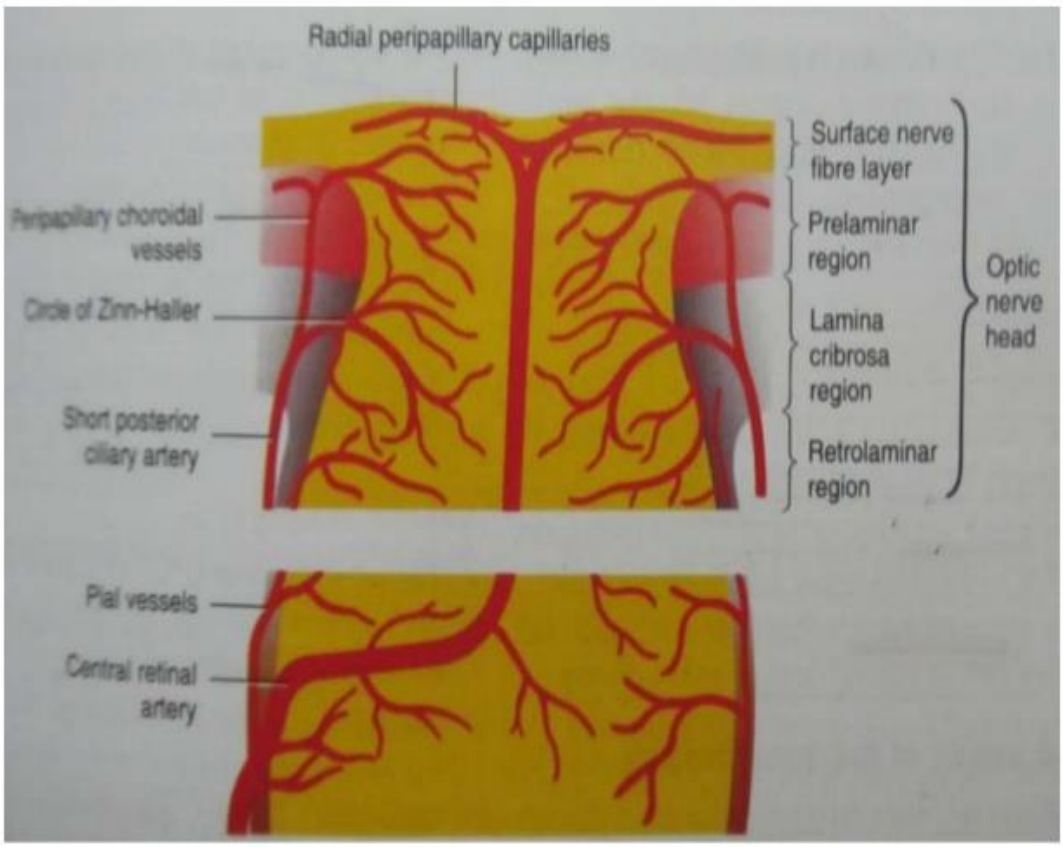
Arterial circle of Willis formed by carotid arterial system of vertebral arterial system.



Blood supply of optic nerve divided into four parts.

A) Intraocular part (Optic nerve)

i. The surface nerve fiber layer mainly supplied by capillaries derived from
ii. Prelaminar region supplied by vessels derived from peripapillary choroidal vessels.
iii. Lamina cribrosa region vessels from posterior choroidal vessels.
iv. Retrolaminar region vessels from centrifugal branches from central retinal artery. Centripetal branches from pial vessels.



B) Intraorbital part:

supplied by two systems

i. Periaxial system → predominant blood supply

ii. Axial system

Periaxial system of vessels derived from 6 branches of internal carotid artery. Ophthalmic artery, long and short posterior cilliary artery, Lacrimal artery, central artery of retina.

Less contribution

Axial system of vessels contains:

- Intra neural branch of central retinal artery.
- Central collateral branch of central retinal artery.
- Central artery of optic nerve.

C) Intra canalicular part

Periaxial system of vessels

D) Intracranial part formed by

Pial system of vessels

Branch of internal carotid artery

Branch of anterior cerebral artery

Branch of ophthalmic artery

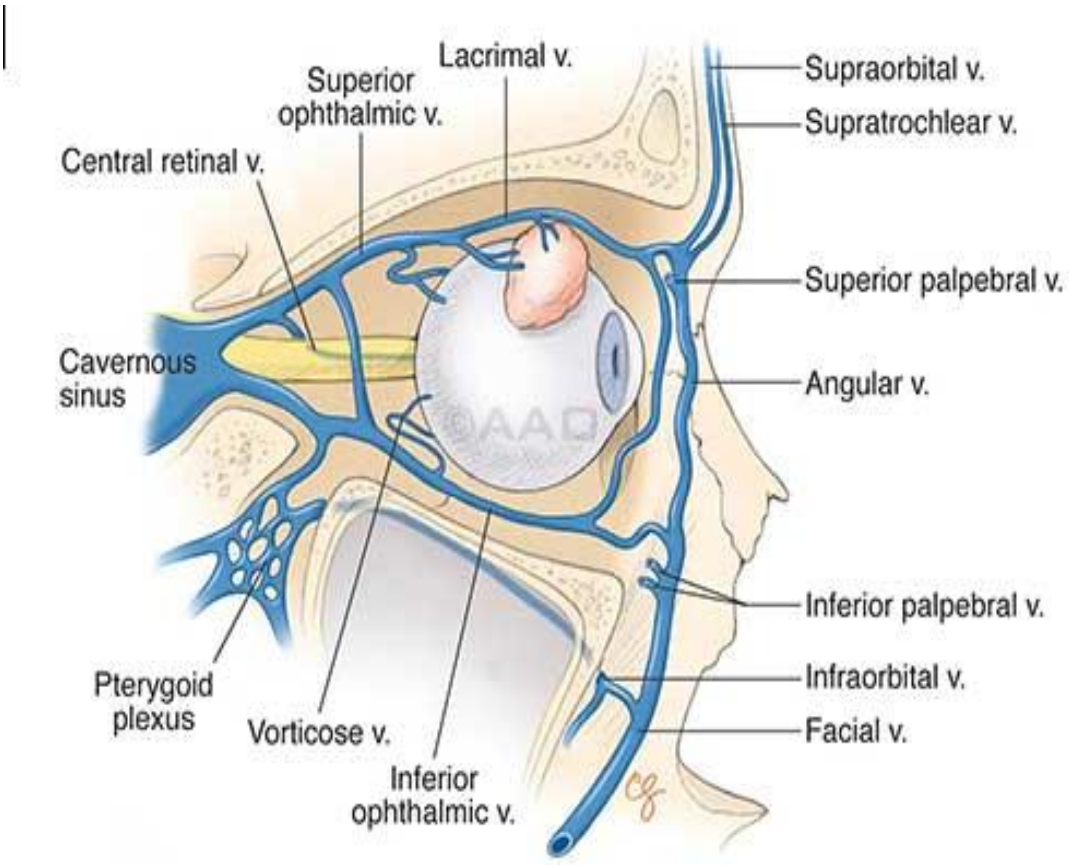
Twigs from anterior communicating artery.

Venous drainage

Optic nerve head → drains through central retinal vein

Orbital part → drains through
peripheral pial plexus,
Central retinal vein

Intracranial part drains through pial plexus which ends in anterior cerebral and basal vein.



Blood supply of optic chiasma:

Arterial supply:

Superior aspect supplied by Branch from anterior cerebral and anterior communicating artery.

Infereior aspect supplied by Branch from internal carotid artery posterior communicating artery, anterior, superior hypophyseal artery.

Venous drainage:

Superior aspect:

drains through superior chiasmal vein drains into anterior cerebral vein

Inferior aspect:

pre infundibular vein draining into basilar vein

Blood supply of optic tract:

Arterial supply

Pial plexus derived from posterior communicating artery, anterior choroidal artery, middle cerebral artery.

Venous drainage through anterior cerebral vein and basal vein.

Blood supply of lateral geniculate body:

Posterior cerebral artery mainly supplies superior homonymous quadrant of retina, Anterior choroidal artery supplies inferior fibers.

Macular fibers supplied by anastomosis from posterior cerebral and anterior choroidal artery.

Optic radiations :

Anterior part mainly supplied by anterior choroidal artery

Middle part supplied by deep optic artery branch of middle cerebral artery. Posterior part supplied by calcarine branches from posterior cerebral artery.

Visual cortex :

Calcarine artery and terminal branch of middle cerebral artery, here middle and posterior cerebral artery anastomosed.

Venous drainage :

Medial aspect – Internal cerebral vein drains in great cerebral vein of Galen and straight sinus.

Superolateral aspect – Inferior cerebral vein drains into cavernous sinus.

EPIDEMIOLOGY :

Serial no.	Type of injury	percentage
1	Motor vehicle accidents	45%
2	bicycle	4%
3	falls	27%
4	assaults	13%
5	others	11%

Road traffic accidents constitutes higher percentage

Young males are commonly affected -79-85%.

Classification :

Mode of injury

1) Direct optic nerve injury
2) Indirect optic nerve injury

Anatomical divisions

1) Anterior optic nerve injury
2) Posterior optic nerve injury

Difference between direct and indirect optic nerve injury:

No.	Direct optic nerve injury	Indirect optic nerve injury
1.	Primary injury to the optic nerve fibers by transaction or infarction Eg. Bullet injury, endoscopic avulsions.	Secondary injury to the optic nerve due to compression as a result of edema and haemorrhage Eg. Blunt trauma to the forehead, and supra orbital ridges.
2	Alters the normal anatomical tissue plane.	Does not alter normal anatomy.
3	Immediate loss of vision occurs.	Delayed visual loss from hours to days.
4	Little likelihood of recovery.	Better after medical and surgical intervention.

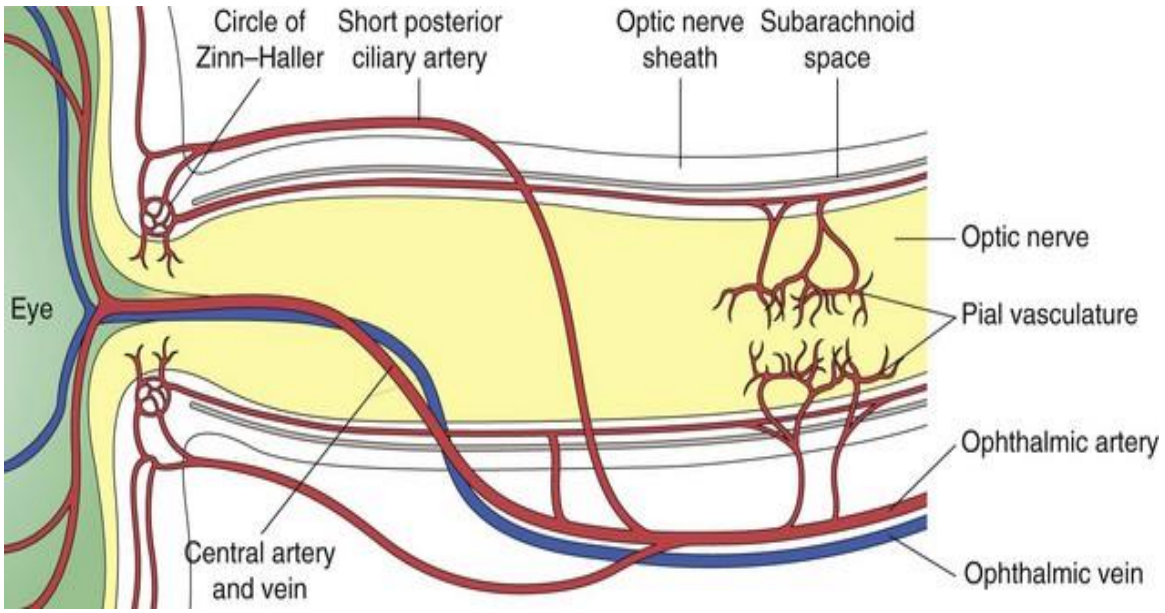
Anatomical classification :

Anterior optic nerve injury :

Central retinal artery and vein enters and exits the optic nerve 8-12mm posterior to the insertion of the nerve into the globe. Injuries anterior to this site are termed anterior optic nerve injury. It causes immediate fundus changes

Posterior optic nerve injury:

Injury posterior to the site of entry of the central retinal artery are called posterior optic nerve injury. Fundus changes occur after 3-5 weeks. The most common site of posterior indirect optic nerve injury is the optic canal next most common site is intracranial part.



Site of traumatic Lesions in visual pathway

I Anterior visual pathway lesio

A) Optic nerve injury:

i) Anterior:

Anterior marginal tear,

Intra orbital part.

ii) Posterior:

Intra orbital part,

Intra canalicular part → commonest site.

Intra cranial part.

B) Opto chiasmal injury

C) Chiasmal injury

II Posterior visual pathway lesions

i) Optic tract and lateral geniculate lesions,

ii) Optic radiation and calcarine cortex lesions.

PATHOLOGY:

Causes of Traumatic Optic Nerve Injury

A) **Primary pathology:**

- Concussion
- Avulsion (Tear)
  - Partial
  - Complete
- Contusion
- Haemorrhage
  - Intraneural
  - Extra neural (sheath)

B) **Secondary pathology:**

- Oedema,
- Ischaemic,
- Microvascular thrombosis,
- Infarction.

Pathogenesis :

Blunt trauma to the superior orbital rim or fronto temporal region,

↓

Compression forces transmitted to the orbital apex and optic canal,

↓

Optic nerve Dura fuses with the periosteum,

↓

Vasculature compromised by compression and contusion,

↓

Swelling exacerbates ischaemia produces compartment syndrome,

↓

Bradykinin, Kaellidin are activated,

↓

Release of oxygen free radicals, intracellular Ca2+ production,
arachidonic acid release,

↓

Peroxidation of lipid cell membranes.

Clinical assessment of optic nerve injuries :

History of closed head injury, fracture involving forehead or supra orbital rim may or may not be associated with loss of consciousness, nausea, vomiting, headache, clear nasal discharge. Complaints of loss of vision ranging from normal to no perception of light, and no complaints of redness, watering, pain, photophobia, bloody discharge, irritation, itching in the injured eye.

Oblique examination of the injured eye :

Minimal swelling may be present over the forehead. Periorbital edema, Ecchymosis, mechanical ptosis, eyelid edema, conjunctival chemosis, haemorrhage, corenal injuries are absent in indirect optic nerve injuries.

Pupillary evaluation :

Direct light reflex:

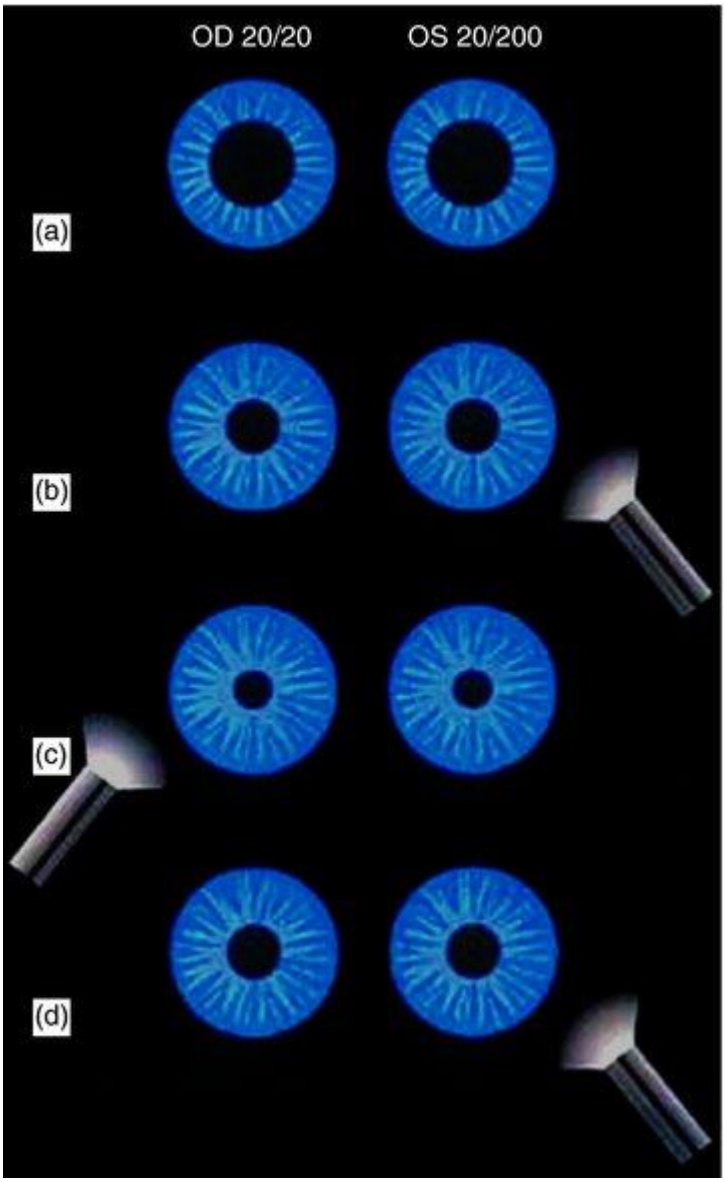
Direct pupillary reflex when light is shone in one eye, there is constriction of ipsilateral pupil in normal individual, which is absent in indirect optic nerve injuries.

Consensual light reflex or indirect light reflex :

When light is shone in one eye, there is constriction of contralateral pupil is known as indirect light reflex. In indirect optic nerve injuries, consensual light reflex present.

Swinging flash-light test :

A bright flash light shone on to one pupil, constriction is noted, then the light is quickly transferred to other pupil and the response is noted. This swinging to and fro flashlight is repeated several times while observing the pupillary response. Normally, both the pupils constrict equally and the pupil to which light is transferred remains tightly constricted.



SWINGING FLASH LIGHT TEST

In case of indirect optic nerve injuries, relative afferent pupillary defect present, here the affected pupil will dilate instead of constriction. (Paradoxical response)

The absence of RAPD indicates there is no indirect optic nerve injury or present bilaterally.

Grades of RAPD :

Grade I : Weak initial constriction and greater redilatation

II : Initial stall and greater redilatation

III: Immediate pupillary dilatation

IV: Immediate pupillary dilatation following prolonged illumination of good eye for 6 seconds

V : Immediate pupillary dilatation with no secondary constriction.

Visual Acuity :

Visual acuity is measured by Snellen,s visual acuity chart done at six meters distance, which may be normal to no light perception (No PL).

Immediate visual loss is common than the delayed visual loss. Transient visual loss due to vasospasm caused by optic nerve concession injury. Secondary visual loss caused by vascular insufficiency mainly affects pial vessels. Delayed visual loss ranging from normal to No PL produced by haemorrhage or increasing edema. Infarction causes permanent visual loss.

In our study, clinical diagnosis confirmed only by visual evoked potential. Visual evoked potential also quantify the optic nerve dysfunction and able to predict the prognosis.

Visual Evoked Potential:

History :

1934 : Adrian, Matthew noticed stimulation of light produced potential changes to the occipital EEG.

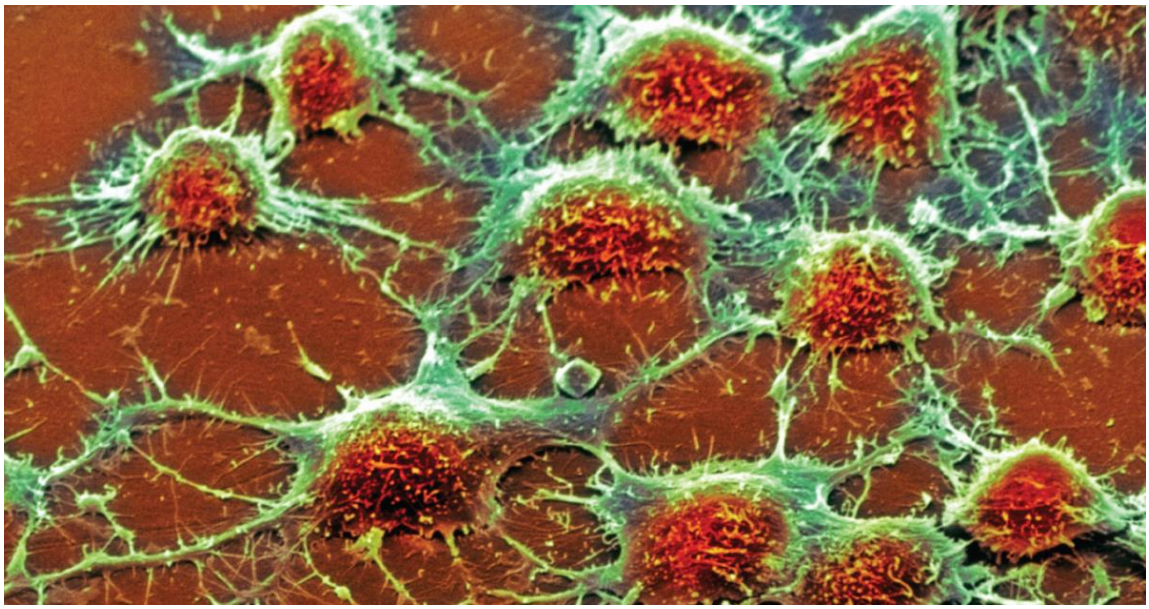
1961 : Hirsch recorded VEP on the occipital lobe.

1965 : Spehlmann first used checkerboard stimulation on human VEP's.

Types of VEP :

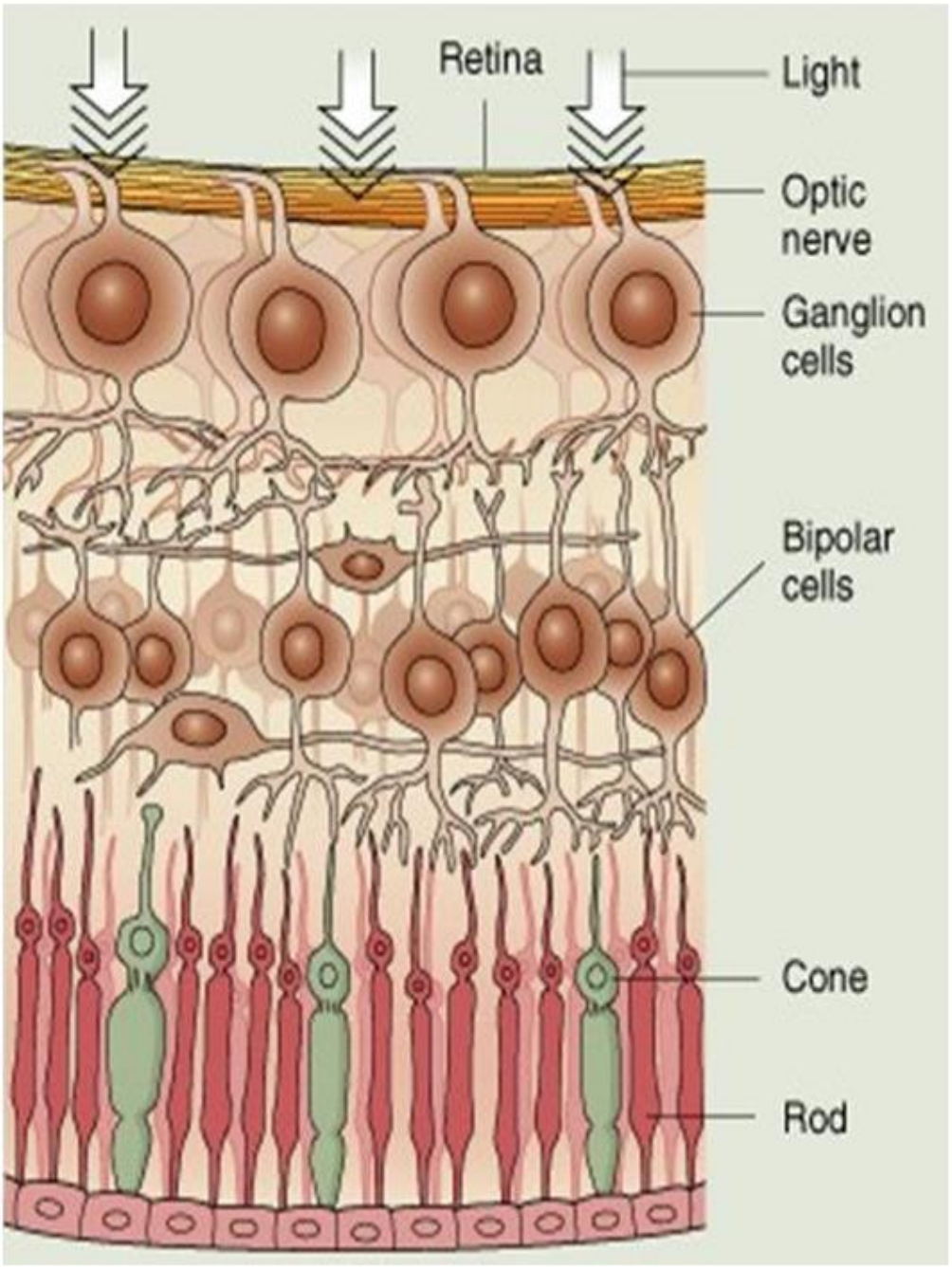
1. Monocular pattern reversal (most common).
2. Sweep visual evoked potential.
3. Binocular visual evoked potential.
4. Chromatic visual evoked potential.
5. Hemi field visual evoked potential.
6. Flash visual evoked potential.
7. LED Goggle visual evoked potential.
8. Motion visual evoked potential.
9. Multifocal visual evoked potential.
10. Multichannel visual evoked potential.
11. Multifrequency visual evoked potential.
12. Stereo elicited VEP.
13. Steady state VEP.

VEP is a electrophysiological test which objectively measures strength and speed of visual signal to the brain, important for immediate identification of indirect optic nerve injuries.



When light falls on the retina electro physiological impulses generated and passed on to the visual cortex via visual pathway recorded by Electro encephalography known as EEG. EEG record taken from occipital lobe is known as VEP.

VEP is the reliable test, detects site of lesions, mainly depends on form sense it gives rough estimate of visual acuity. It assess the functional state of the visual system beyond the retinal ganglion cells.

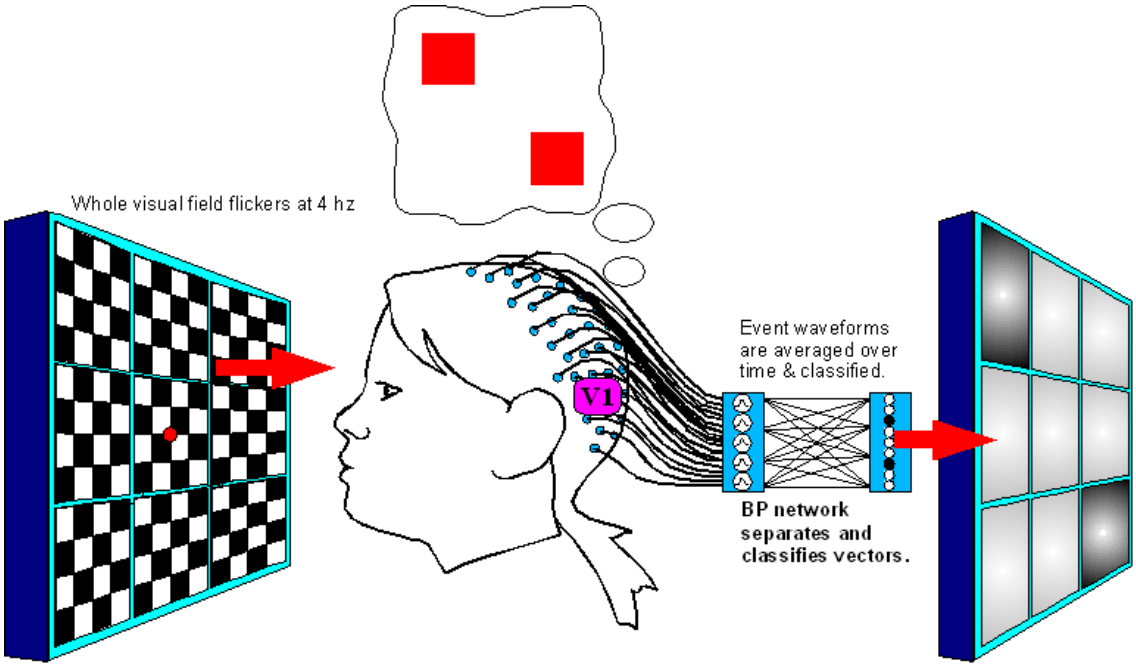


Prerequisites for VEP

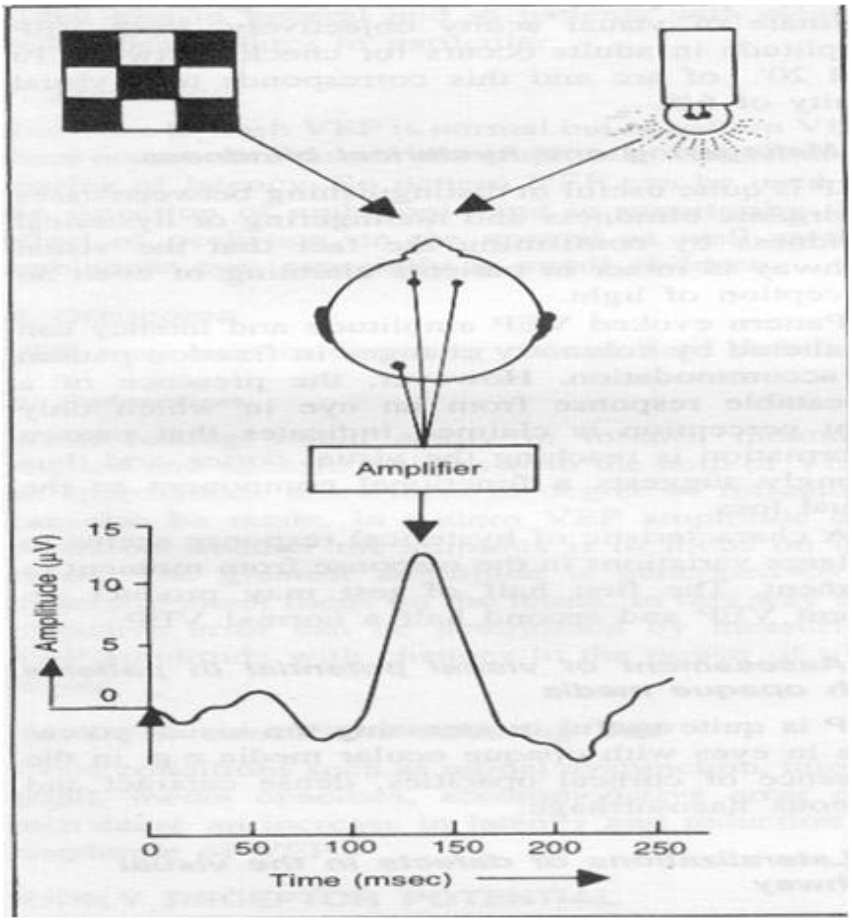
- ✓ Patient should be conscious, able to seat comfortable in front of the monitor 0.75 – 1.5meter.
- ✓ Patient should not apply oil.
- ✓ Each eye tested separately.
- ✓ Eye glasses to be worn.
- ✓ Other eye should be patched.
- ✓ Gage at the centre of the monitor.

Equipment for recording VEP :

- ✓ Visual stimulus producing device,
- ✓ Suitable scalp electrode,
- ✓ Amplifier,
- ✓ Computer and read out systems.



Types of VEP recording:



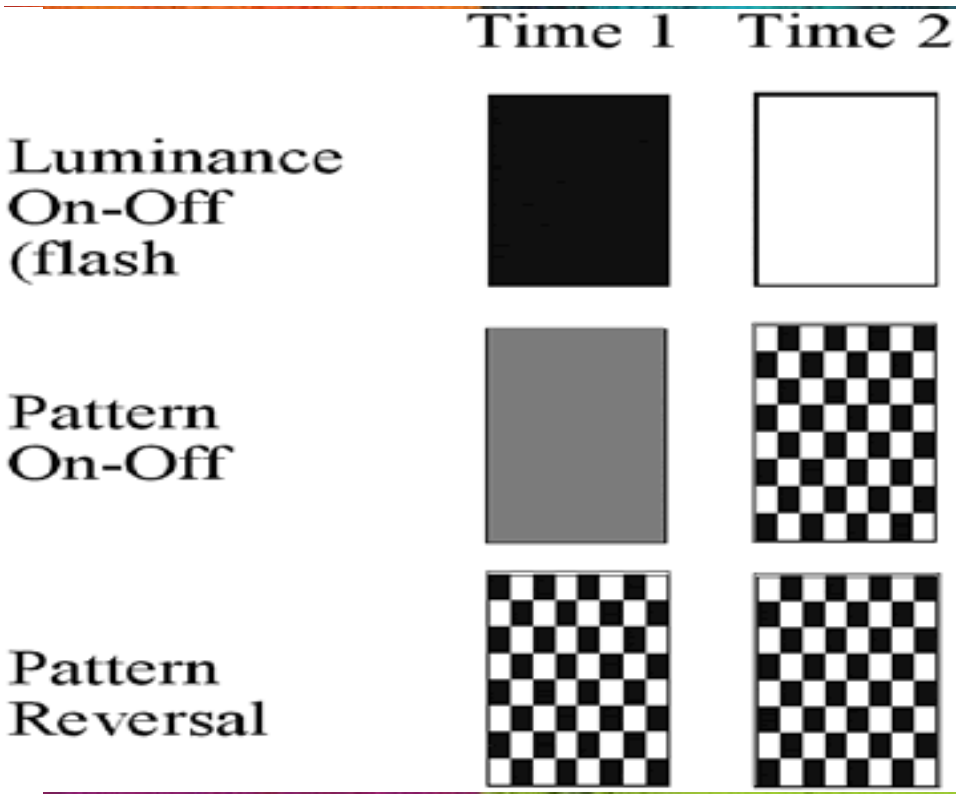
1. Flash VEP

A calibrated intense diffuse light or stimulus that releases flashes from a shutter 1-5 times / sec. It indicates light perceived by the visual cortex not affected by the opacities.

2. Pattern VEP

Patterned stimulus displayed on a TV screen in the form of black and white chess board squares , its size can be adjusted known as checker board pattern.

Two types :



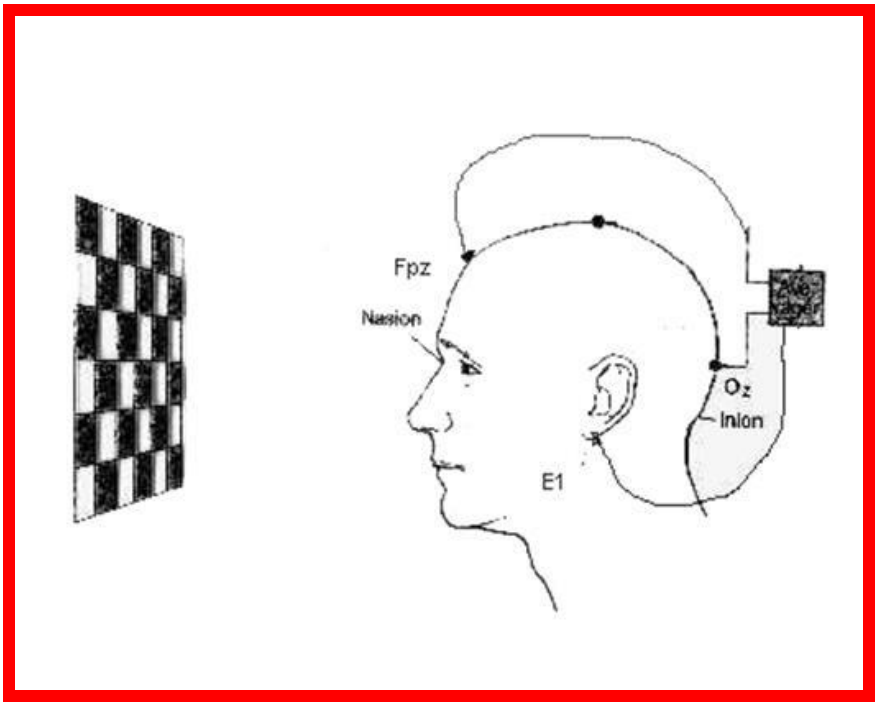
Pattern appearance VEP : a black and white checker board presented in on off sequence.

Pattern reversal VEP : The pattern stimulus changed black squares turns white and white squares turns black alternately.

VEP electrode placement :

Recording electrodes are made either silver chloride or gold disc.

One electrode is placed 2.5cm above the inion and a reference electrode placed at Fz. Two additional electrodes placed 2.5cm to the right and left of Oz.



Active electrode

Midline occiput (MO)-oz

Reference electrode

Vertex Cz

Ground electrode

Forehead Fpz

Recording conditions

Band pass:1-300Hz

Analysis time:250ms

Number of epocs: minimum 100.

Electrode impedence: <5Ω

Stimulation patterns:

- Black and white checker board.
- **Size of the checks:**14 x 16 minutes.
- Size and distance from the monitor should produce a visual angle of 10-20˚.
- **Contrast:** 50-80%.
- **Mean luminance:**

  Central field -50cd/m^2.

  Background 20-40cd/m^2.

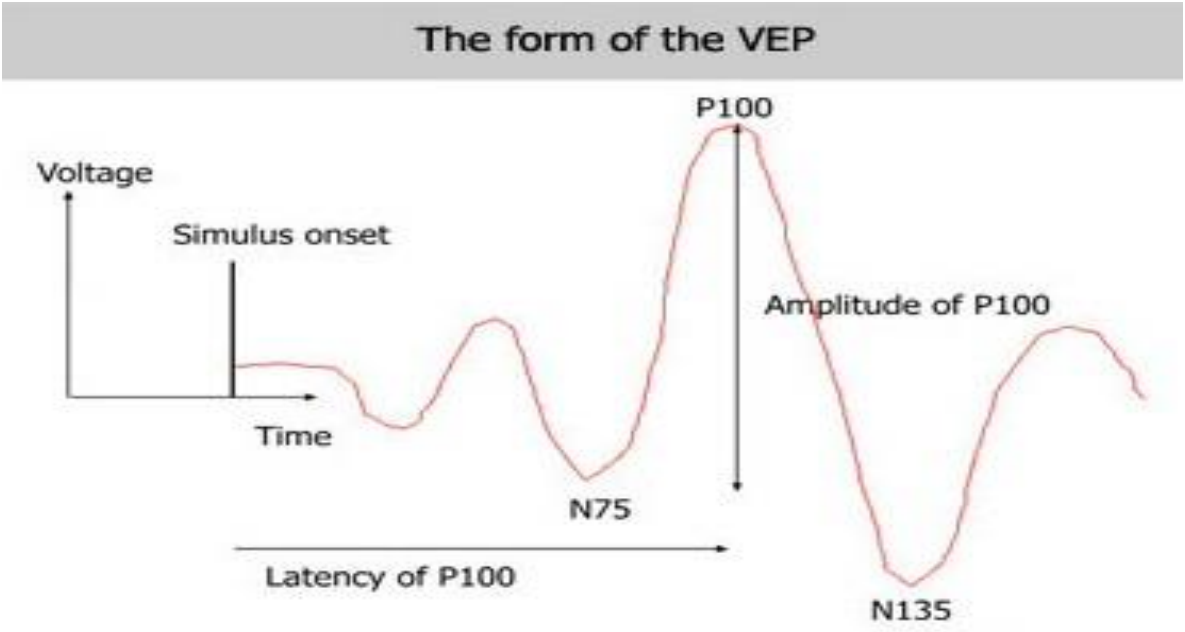
VEP wave forms :

VEP shows three wave forms

i) primary positive wave - peak latency of 100ms.upper limit of normal 117-120milliseconds.

ii) Two negative waves- peaks at N75&N135.

iii) Inter eye latency difference for P100 should be less than 6-7 milliseconds.



VEP terminologies:

Amplitude of VEP:

Height of the potential P100 wave, measured in microvolts, indicates amount of electrical energy reaching visual cortex.primarily affected in ischemic disorders.

Latency of VEP:

Time from stimulus onset to peak of the response, measured in milliseconds, indicates time taken for electrical signal to reach visual cortex,seen in demyelinating disorders.

Bizzare wave forms :

both amplitude and latency is predominantly affected in compressive disorders and optic nerve injuries.

Indications :

1. Optic nerve disease.
2. Inherited retinal dystrophies.
3. Vascular diseases.
4. Opaque media or trauma.
5. Unexplained visual loss.
6. Infant with questionable vision.
7. Toxic and nutritional eye disease.
8. Glaucoma.
9. Suspected intracranial lesions.
10. Malingering and hysterical blindness.
11. High Refractive errors.
12. Amblyopia.

Factors influencing VEP

1. **Stimulus :**

   In transient response increases in amplitude with decrease in the size of checks, reaching a peak when the check subtends about 15 arc at the eye.

2. **Position of electrodes:**

   Scalp influences the character of normal VEP response.

3. **Age and Sex :**

   Females have larger responses than males.

4. **Attention of the patient to stimulus:**

Large macular representation on occipital cortex, response mainly divided from the central few degrees of the retina.

5. **Effect of diseases on VEP:**

1.**Optic neuritis**:

Amplitude of VEP wave form become normal, following resolution latency almost always prolonged in permanent damage.

2. **Multiple sclerosis:**

Delayed latency noted.

3. **Compressive optic nerve lesions-**

Reduction in amplitude without much change in the latency.

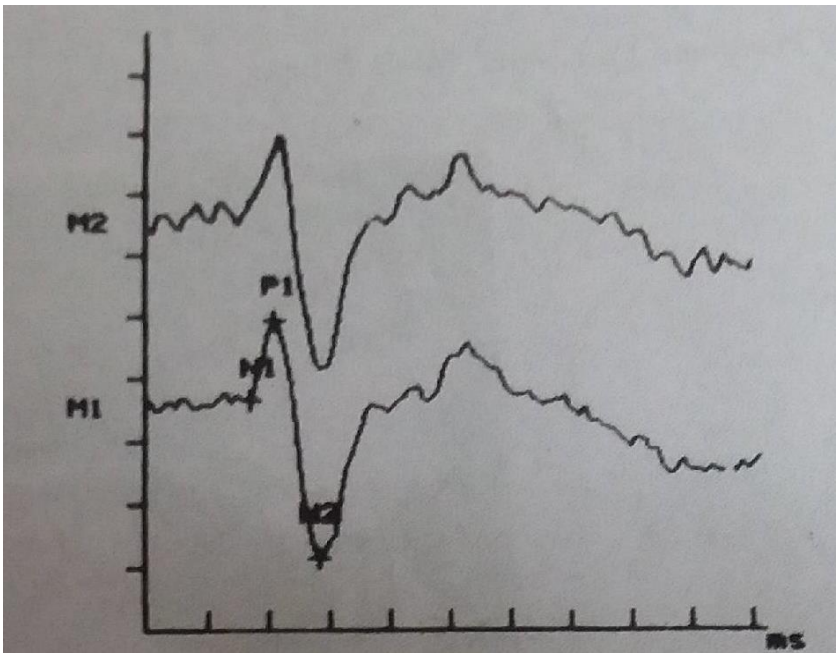
4**. During orbital or neurosurgical procedures**

Continuous record of optic nerve function by using VEP preventing inadvertent damage of nerve during surgical manipulation.

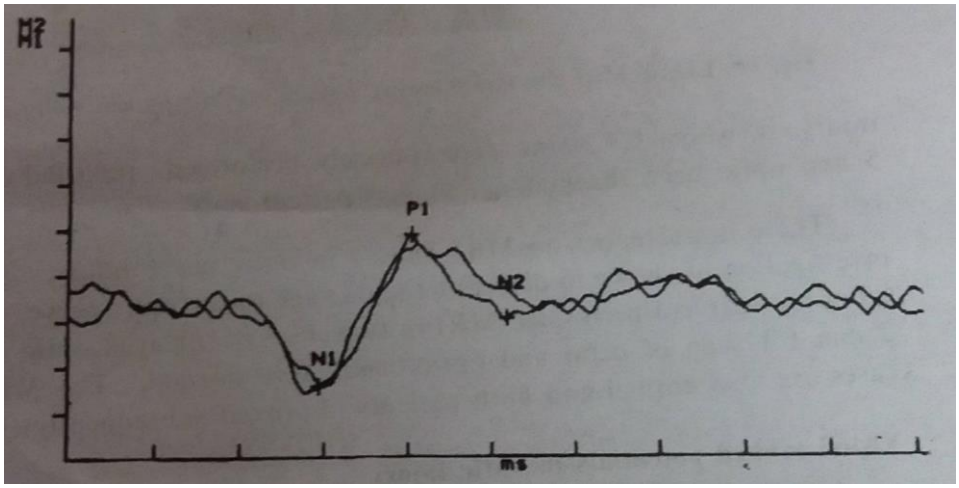
5.**Traumatic optic nerve injuries**

Mainly delayed latency period of P100 is more predominant than decreased amplitude indicates partial loss of optic nerve fibers.

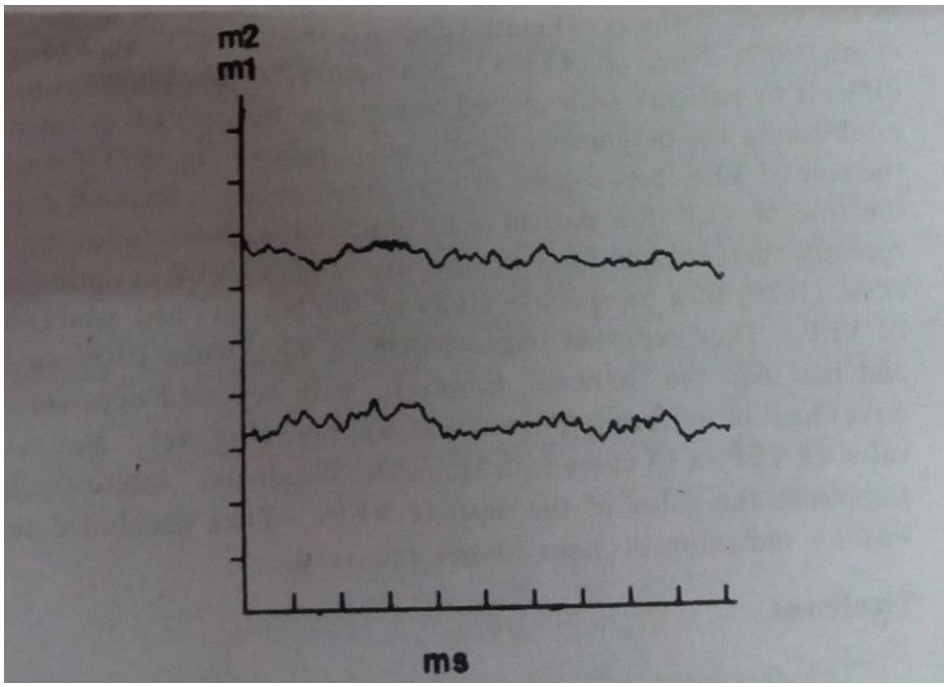
Absence of VEP wave or no response denotes total loss of optic nerve fibers, are the indicator of poor visual recovery.



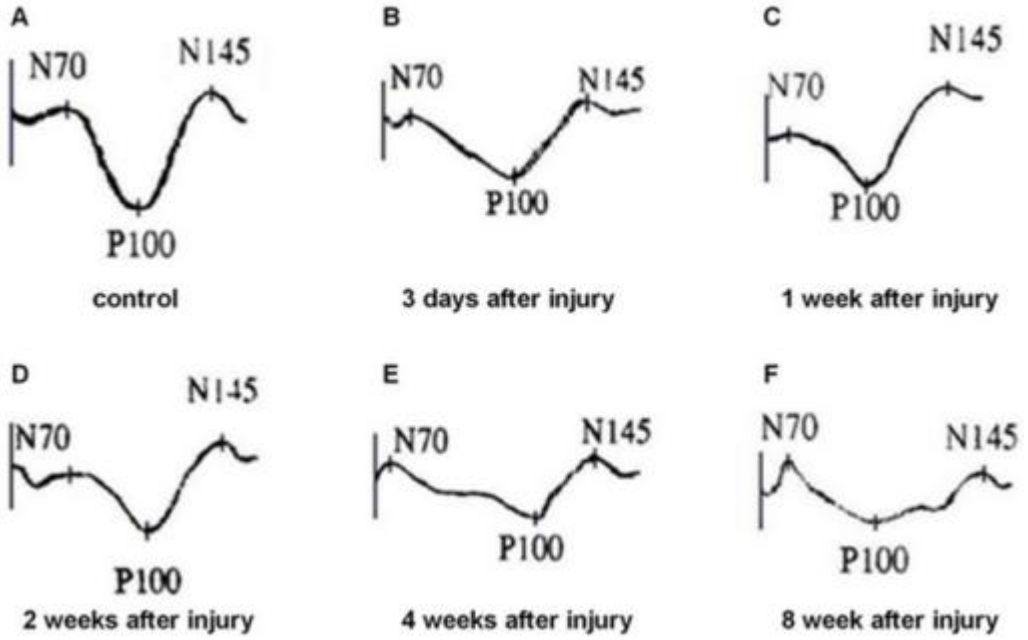
VEP SHOWING NORMAL P1 WAVE



VEP SHOWING DELAYED P1 LATENCY IN A PATIENT WITH OPTIC NERVE INJURY



VEP IN A PATIENT WITH OPTIC NERVE INJURY SHOWING NO P1 WAVE



Normative values of visual evoked potential:

Parameter	Range	Mean ± SD	Range	Mean ± SD
	15 checks		31 checks	
P 60 latency	50.0 - 75.0	60.9 ± 4.2	44.5 - 67.0	56.1 ± 3.5
N70 latency	63.5 - 87.5	75.5 ± 4.1	60.0 - 87.5	70.8 ± 3.7
N70 amplitude	1.0 - 18.2	5.1 ± 3.1	0.7 - 14.5	3.9 ± 2.2
P100 latency	83.5 - 107.5	98.1 ± 4.4	81.5 - 107.0	94.7 ± 5.0
P100 amplitude	1.1 - 38.1	9.9 ± 5.9	1.9 - 29.9	8.7 ± 4.7

Colour Vision:

Colour vision tested by pseudoisochromatic colour plates.

In optic nerve injuries red object looks like black, brown or orange, described as ‘faded’. Severely injured cannot be identified at all.

Field defect :

Visual fields are tested by Bjerrum’s method, if the visual acuity is > 2/60 following injury. Central, paracentral, centrocecal, hemianopic defects, altitudinal defects and generalized field constriction are noted.

Intraocular pressure:

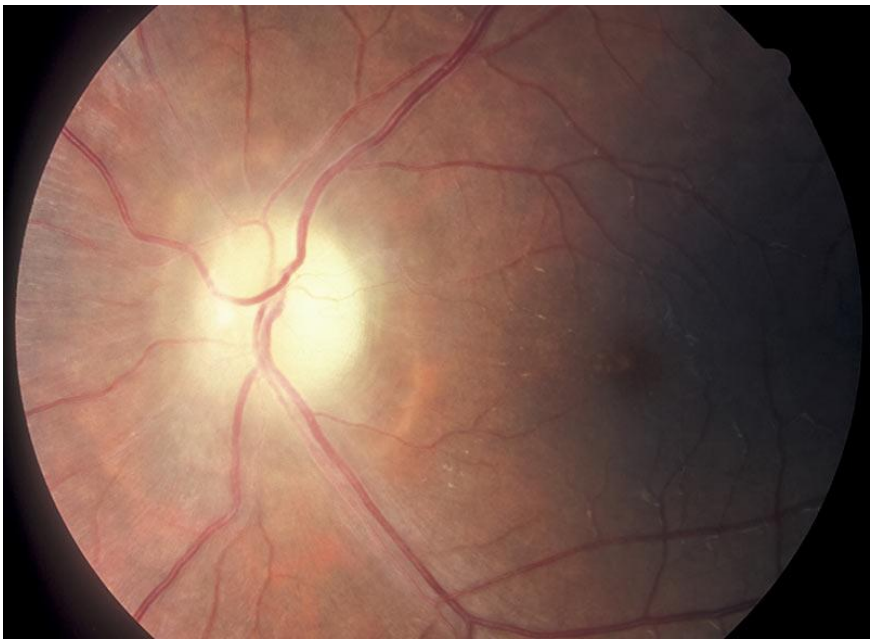
Intra ocular pressure measured by Schiotz”s tonometer,which is very useful for bedside evaluvation.normally 16-20mmg present,if it is more than 20mmHg indicates increased intra ocular preesure caused by undue pressure over the over the eyeball.the opposite eye acts as a control.

Fundus examination :

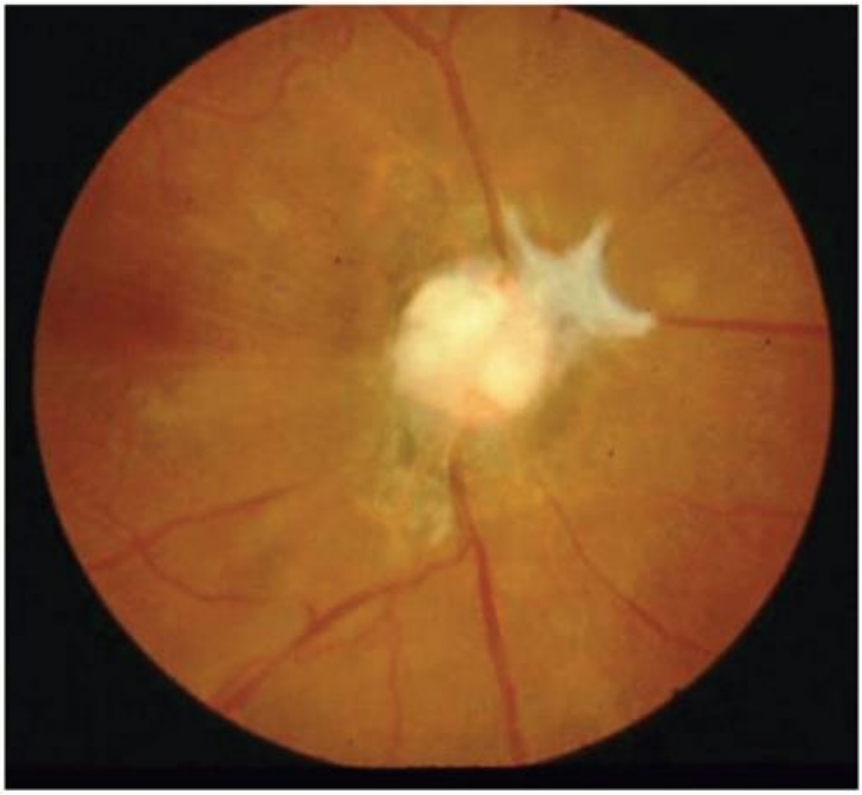
Fundus examination done by direct ophthalmoscopy if possible slit lamp biomicroscopy with 90D lens.

Retinal haemorrhage, irregular disc margin occurs, which is disappear and leave pigmented scar later. Pale disc with grey retina and thinned blood vessels, cherry red spot of macula are also noted. Optic atrophy occurs after 4-6 weeks.

OPTIC NERVE AVULSION



OPTIC NERVE ATROPHY



OLD POST TRAUMATIC OPTIC ATROPHY WITH GLIOSIS

Neuroimaging :

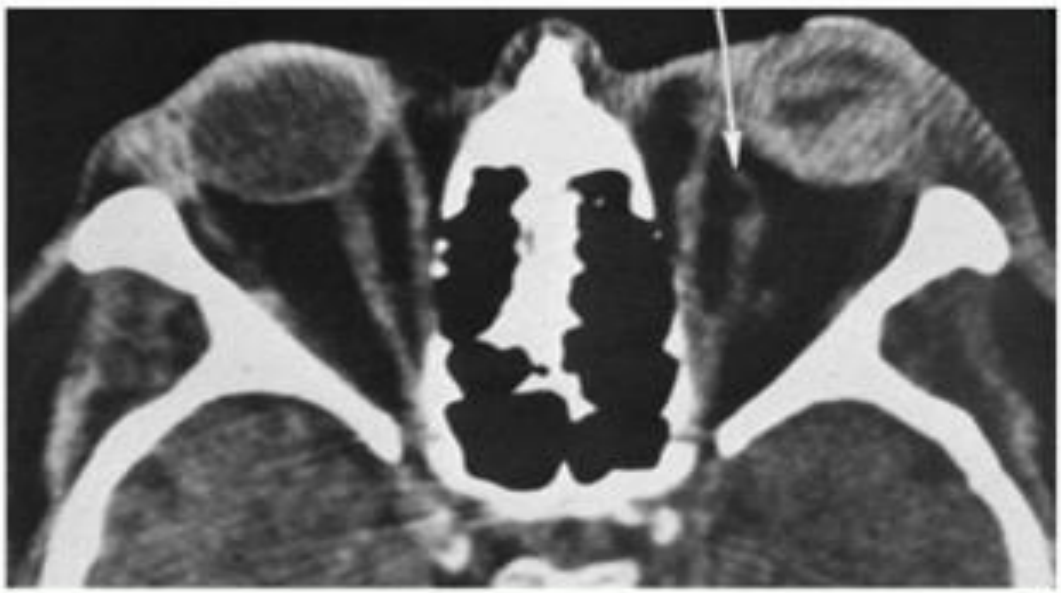
X ray

Soft tissue opacities in paranasal sinuses and air fluid level indirectly indicates fracture of the anterior cranial fossa.

CT Scan

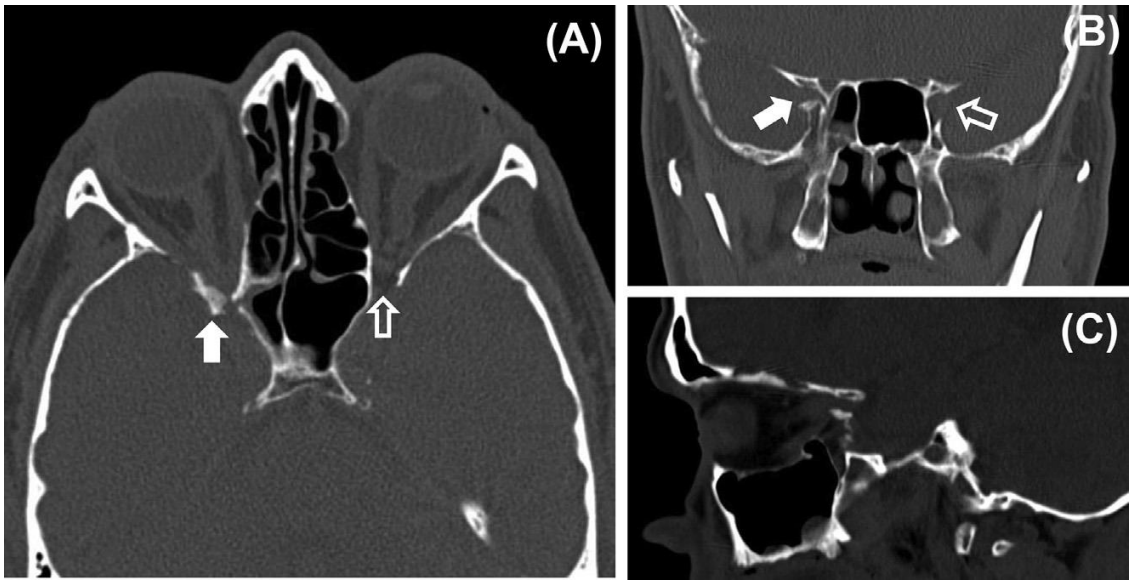
CT scan reveals sphenoid and ethmoidal haemorhage, Metallic foreign body, proptosis and soft tissue injuries.

CT scan is useful in diagnosing fractures of optic canal and displaced bony fragment which impinge on the optic nerve. It also helps to diagnose optic nerve sheath haematoma and subperiosteal haematoma.

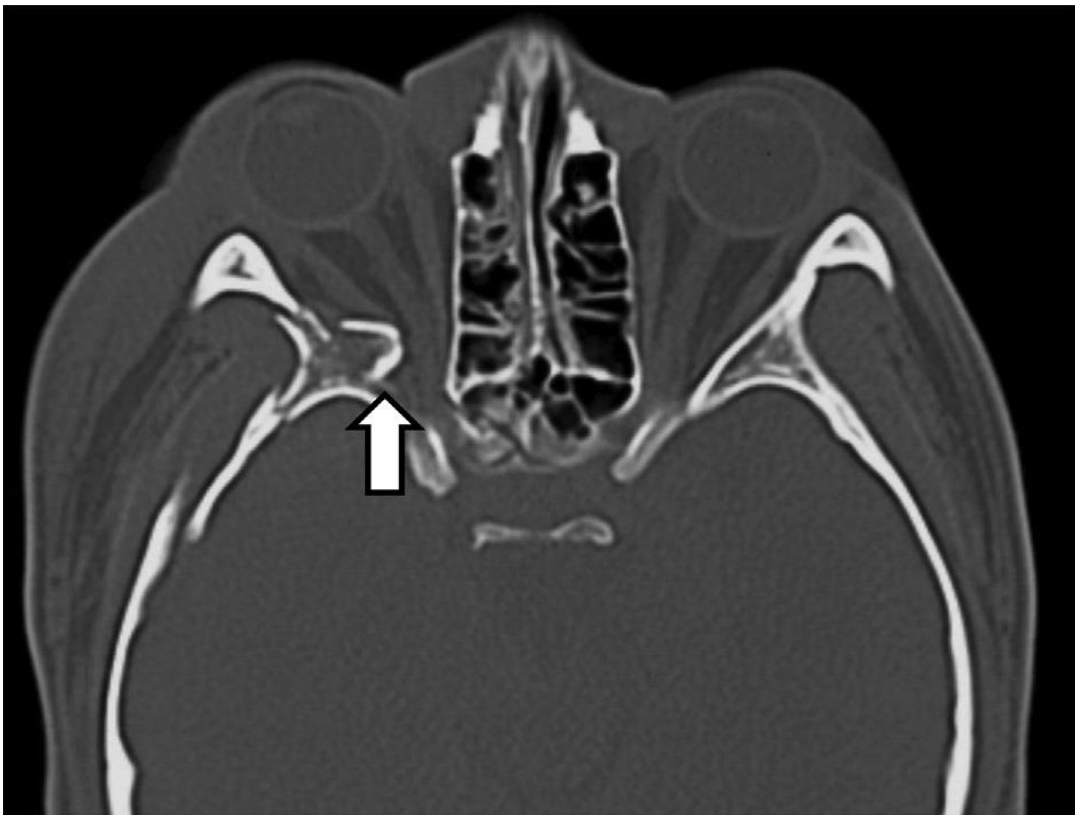


CT IMAGE OF OPTIC NERVE AVULSION

OPTIC NERVE SHEATH HEMATOMA



OPTIC CANAL FRACTURE AND OPTIC NERVE COMPRESSION



LATERAL ORBITAL WALL FRACTURE WITH BONY IMPINGEMENT OF THE OPTIC NERVE

MRI scan :

MRI scan is the investigation of choice for detecting indirect optic nerve injuries. MRI rules out the inflammatory, infiltrative, haemorrhage that involves neurovascular structure, before doing MRI rule out intra ocular metallic foreign body.

POST TRAUMATIC OPTIC NERVE SHEATH EDEMA

POST TRAUMATIC OPTIC NERVE SHEATH EFFACEMENT IN RIGHT ORBIT

Diagnosis:

Indirect traumatic nerve injury clinically diagnosed only on pupillary examination by direct light reflex which is absent here and consensual or indirect light reflex is present. Swinging flash light test detects the RAPD,presence of RAPD gives clinical diagnosis of indirect optic nerve injury. Anterior segment examination is normal except relative afferent pupillary defect.

Indirect optic nerve injury diagnosed on pupillary finding can be confirmed possibly only by VEP.

Differential Diagnosis:

1. Optic nerve sheath hematoma
2. Orbital hematoma
3. Subperiosteal hematoma
4. Coincident optic neuropathies
5. Compression by tumor or aneurysm
6. Optic nerve Inflammation
7. Orbital Inflammation
8. Sinusitis with orbital involvement
9. Ischemic optic neuropathy
10. Optic neuritis.

Treatment :

Four methods are available

1. Conservative management
2. Medical management
3. Surgical management
4. combined approach

Conservative management :

Conservative management useful in those who are contraindicated for high dose steroid therapy. It includes diabetes mellitus, systemic hypertension, seriously ill patients, cardiac patients, peptic ulcer disease.

Medical Management :

The international optic nerve trauma study was done to assess the benefit of high dose steroids and surgery in the treatment of indirect traumatic optic neuropathy.

steroids used in various doses as low [<100mg], high [500-200mg], very high [2000-5399mg] and mega dose [more than 5400 mg]. These are based on daily initial dose of methlprednisolone.

In our hospital we are following high dose steroid therapy according to **Optic Neuritis Treatment Trial (ONTT).**

Traumatic Optic neuritis treatment trail

This regimen is based on **Optic Neuritis Treatment Trial**. The treatment used for optic neuritis is applied for the treatment of indirect traumatic optic nerve injuries.

Intravenous methylprednistone 1 gm OD x 3 days followed by 1mg/kg/per day of Oral prednisolone tablet for 11 days and then taper.

Patients are also administered histamine receptor blockers and calcium supplement along with steroid. Oral prednisolone is rapidly tapered and stopped.

Mega dose methylprednisolone regimen

This regimen is based on **second national spinal cord injury trial in 1990 (NASCIS II)**[5]. This trial was done for patients with acute spinal cord injury which showed good improvement in those patients. In this regimen injection IV methylprednisolone is administered with an initial dose of 30mg/kg. This is followed by a continuous infusion at the dose of 5.4 mg/kg/hr for 48 hours. Higher death rates are reported in this mega dose steroids. This is the major disadvantage of this regimen.

Precautions during steroid therapy

During treatment with high dose steroids it is necessary to monitor blood pressure, blood sugar and cardiac status. High dose steroids can induce cardiac arrhythmias, so it should be given under strict cardiac monitoring.

STUDIES SUPPORTING STEROID THERAPY

The **second national spinal cord injury study (NASCISII)**,was a multicentric, randomized placebo-controlled, double blind study conducted in patients with acute spinal cord injury.

The **Corticosteroid Randomisation after significant Head injury** (**CRASH)** shows effectiveness and safety of steroids in traumatic optical nerve injuries.

Mechanism of action of steroids:

1. Decreases intra neural and extra neural edema-relieving compression of the nerve fibres.
2. Steroids reduce the vasospasm and limit the contusion necrosis of the nerve and block the neuronal death through the inhibition of free radicals.
3. Antioxidant properties of steroid produces neuroprotective effert and inhibits free radial induced lipid peroxidation.

Complications of steroids:

1.Gastro intestinal bleed

2.Wound infections

3. Pneumonia

4.Pancreatitis

5.Sepsis

6.Acute psychosis.

Surgical Management :

Surgical management of the optic nerve done via transcranial transethmoidal or endonasal trans ethmoidal approaches.

Guidelines :

i) Surgery should not be done in unconscious patient.

ii) Immediate vision loss with non reacting pupil surgery is contraindicated.

iii) Delayed type of vision loss, surgical intervention gives possible improvement.

iv) If the time of onset of vision loss not able to determined, wait and watch for few days because spontaneous recovery occurs in some cases.

Intranasal endoscopic approach is little advantageous because of the proximity of optic nerve. The aim of optic canal decompression is to relieve the compression and improve the vascular supply to the intracanalicular part of the optic nerve. Since the optic nerve is confined within the bony space of optic canal, decompression tend to reverse its compression in this space.

The criteria for surgical decompression includes :

1. Removing minimum 50% of osseous canal circumference.
2. The bone along entire canal length to be removed.
3. Longitudinal incision of entire dural sheath along with annulus of zinn.

Various surgical approaches are used for optic nerve decompression. It can be performed by transcranial or extracranial approaches. Intracanalicular and intracranial portions of optic nerve can be decompressed by frontal craniotomy. This procedure is associated with minimal morbidity and good visual recovery. Systemic steroids are given preoperatively in loading dose and continued postoperatively every 8 hours for next 24 hours.

Surgery is useful for treatment of patients with indirect optic nerve injury. In case of fractures involving optic canal, reduction of bone fragments impinging on optic nerve is done. Surgery is useful for the evacuation of intraoptic nerve sheath hematoma. It can also be done by using lateral or medial orbitotomy according to location of hematoma. Lateral canthotomy with cantholysis is done in patients with orbital hemorrhage. This causes orbital content expansion and relieve compression on the optic nerve.

Combined therapy :

Both Medical and Surgical Management

1. All cases high resolution CT scan should be done immediately.
2. VEP should be done as soon as possible and repeated every 2-3 days to assess clinical and electrophysiological improvement.
3. Optic nerve injury once diagnosed immediately start cortico steroids in acute stage.
4. If no improvement after 2-3 weeks of conservative treatment indicates very bad prognosis and the patient give option for surgical approach.
5. Patients with delayed visual deterioration give high dose of cortico steroids, repeat VEP, and consider decompression in case of no visual improvement or deterioration on steroids.

Follow up:

1. Visual acuity
2. RAPD
3. VEP
4. Colour vision
5. Fields
6. Fundus examination

Patient should be examined pertaining to the above parameters in every visit and they are followed upto 2 months.

Prognosis :

Good prognostic factors :

1.Indirect trauma

2.No history of loss of consciousness

3.Good initial visual acuity

4.Lower grades of RAPD

5.Signs of visual recovery within 48 hours

6.Absence of optic canal fracture

Poor prognostic factors:

1.History of loss of consciousness

2.No improvement in vision after 48 hours

3.Evidence of optic canal fracture

4.Grade IV, V of RAPD

5.Absence of VEP response

AIMS AND OBJECTIVES OF THE STUDY:

To evaluate the role of visual evoked potential in patients with indirect optic nerve injuries.

To study about the following factors in patients with indirect traumatic optic nerve injuries and assessment of final visual outcome

- Visual acuity at the time of presentation
- Pupillary examination - RAPD
- Visual evoked potential
- Color vision defect and types.
- Field defect at presentation
- Fundus examination

Study design:

A hospital based prospective, observational study

Study centre:

Department of Ophthalmology, Department of Neuromedicine, Madurai Medical College, Government Rajaji Hospital, Madurai which is a tertiary care centre.

Duration of study:

6 months (Jan 2017 – June 2017)

Study population:

60 patients

Case selection:

Patients are selected among those admitted in trauma ward with head injury, ophthalmic examination are done and those patients diagnosed as indirect optic nerve injuries were selected for study on the basis of inclusion and exclusion criteria.

Inclusion criteria:

1. Patients with head injury irrespective of initial visual acuity at the time of presentation.
2. Age more than 20 years (Male and female patients).
3. Diagnosis of indirect traumatic optic nerve injury within 8-12hours of trauma.
4. Normal or near normal field of vision.

Exclusion criteria:

1. Rupture globe
2. History of loss of consciousness for more than 24 hours
3. Patients with concurrent traumatic brain parenchymal injury.
4. History of seizures
5. History of underlying neurological disorders, neurotoxic drugs.
6. History of diabetes mellitus, systemic hypertension, cardiac disorders, psychiatric illness, alcoholism
7. Mature cataract , glaucoma and other ocular optic nerve disc diseases.
8. Patients with congenital ocular diseases (Eg.Retinitis pigmentosa)
9. Optic nerve avulsion
10. Optic neuritis (all types), optic atrophy, ischemic optic neuropathy.

11. Past history of eye surgery with retro bulbar block.
12. Prior neurosurgical intervention
13. Patients not willing for regular follow-up.

Materials and Methods:

Prospective, observational study to detect the subclinical findings in indirect traumatic optic nerve injury patients using visual evoked potential and to start immediate treatment for better final visual outcome.

Procedure:

Patients satisfying both inclusion and exclusion criteria are selected.

Explain about the study and procedure and get informed consent.

i. Initial Visual acuity is recorded by Snellen's chart.
ii. Anterior segment examination by torch light and slit lamp biomicroscopy.
iii. Pupillary examination (both direct, consensual and swinging flash light test) by pupilloscope.
iv. VEP be performed in sitting position.
v. Colour vision tested by psuedoisochromatic ishihara's color vision chart
vi. Visual fields by Bjerrum's method.
vii. Tension by Schiotz's tonometer
viii. Fundoscopy by direct ophthalmoscope or by using +90D lens with slit lamp are recorded.
ix. Patients are treated with drug.

STATISTICAL ANALYSIS:

The information collected regarding all the selected random samples were recorded. Data analysis was done with the help of computer by using SPSS 16 software and Sigma stat 3.5 version. Using this software mean , Standard deviation and ‘p’ value were calculated through student ‘t’ test, Chi square test, person correlation and p value of <0.05 was taken as significant.

OBSERVATION AND ANALYSIS:

TABLE 1: AGE DISTRIBUTION

Age Distribution	No.of cases	Percentage
< 25	12	20.0
26 - 30	19	31.7
31 - 35	21	35.0
> 35	8	13.3
Total	60	100.0
Mean	30.2	
Range	19-42	

The average age of the patients was 30 years (Range:19-42 years)

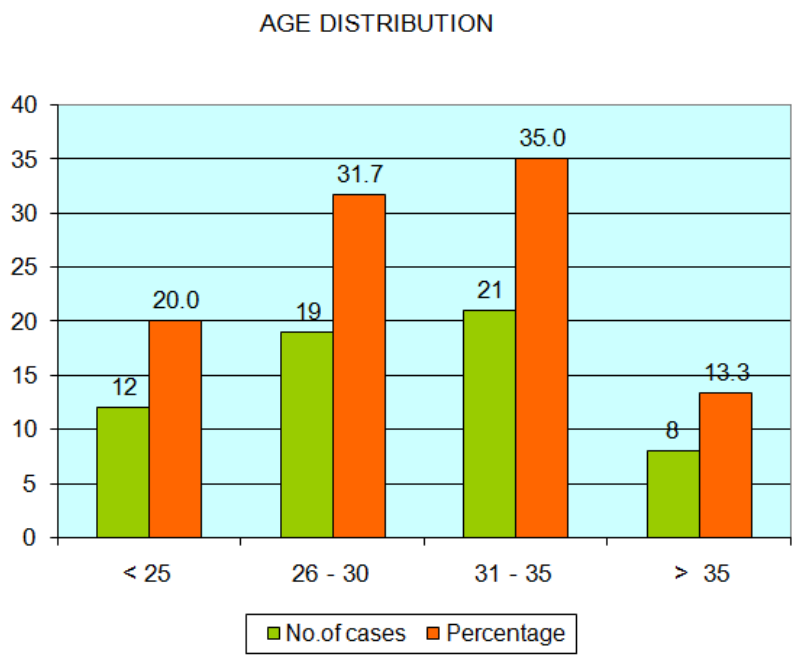


TABLE 2: SEX DISTRIBUTION

Sex	No.of cases	Percentage
Male	45	75.0
Female	15	25.0
Total	60	100.0
Gender ratio	3 : 1	

Our study showed a clear male preponderance as expected.

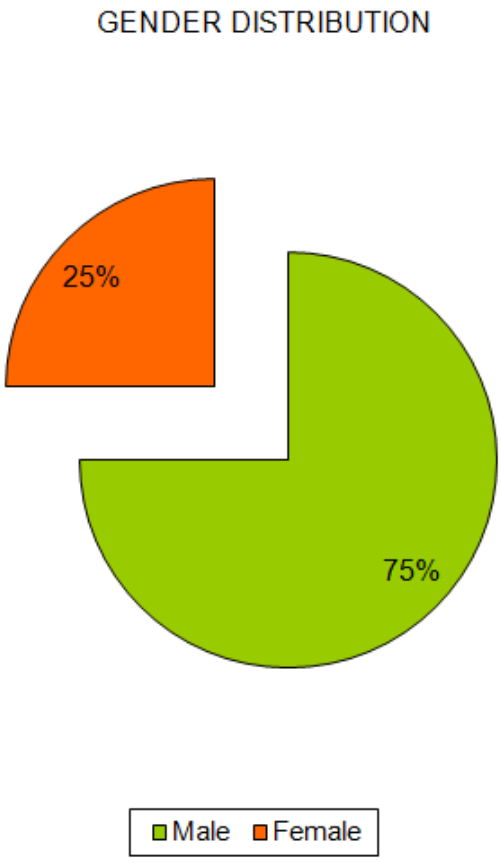


TABLE 3: LATERALITY

Eye	No.of cases	Percentage
Right	44	73.3
Left	16	26.7
Total	60	100.0

Right eye was more frequently involved **p value <0.001**

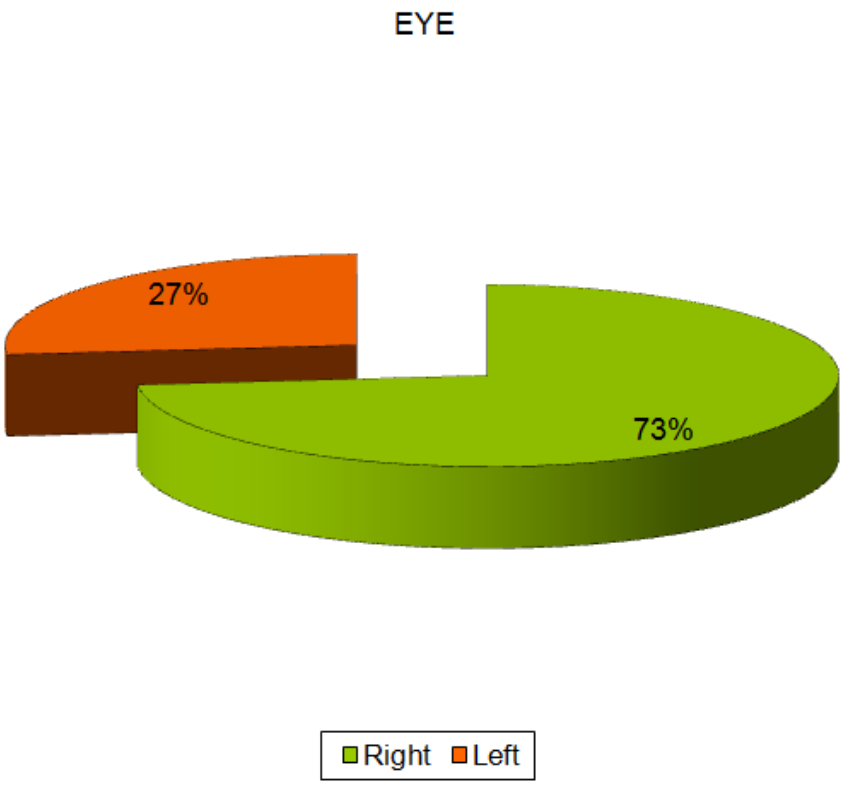
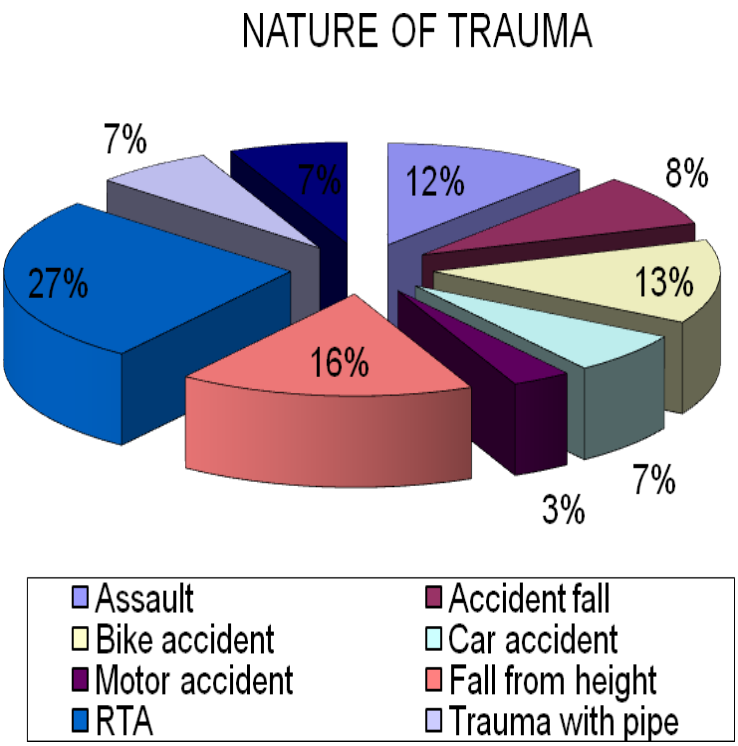


TABLE 4: NATURE OF TRAUMA

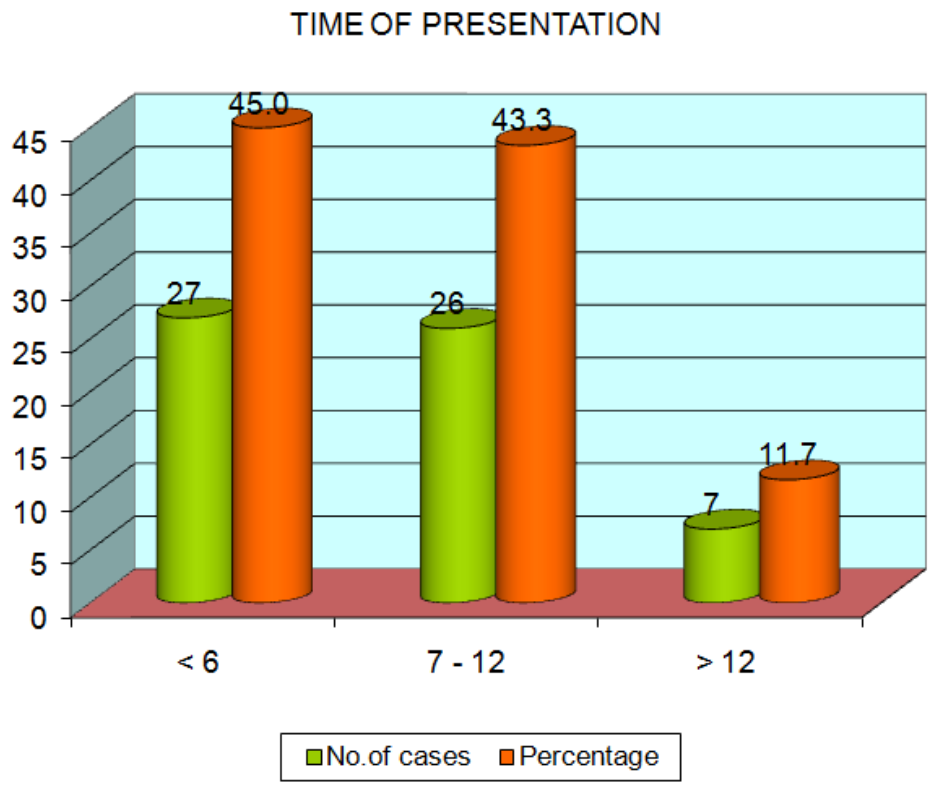
Nature of Trauma	No. of cases	Percentage
Assault	7	11.7
Accident fall	5	8.3
Bike accident	8	13.3
Car accident	4	6.7
Motor accident	2	3.3
Fall from height	10	16.7
RTA	16	26.7
Trauma with pipe	4	6.7
Trauma with stone	4	6.7
Total	60	100.0



Road traffic accidents were the commonest cause amongst which two wheeler accidents constituted the major portion. Fall from height was the second most common cause. Assault, accidental fall and trauma with pipe contributed to the remaining proportion of indirect optic nerve injuries.

TABLE 5: TIME OF PRESENTATION

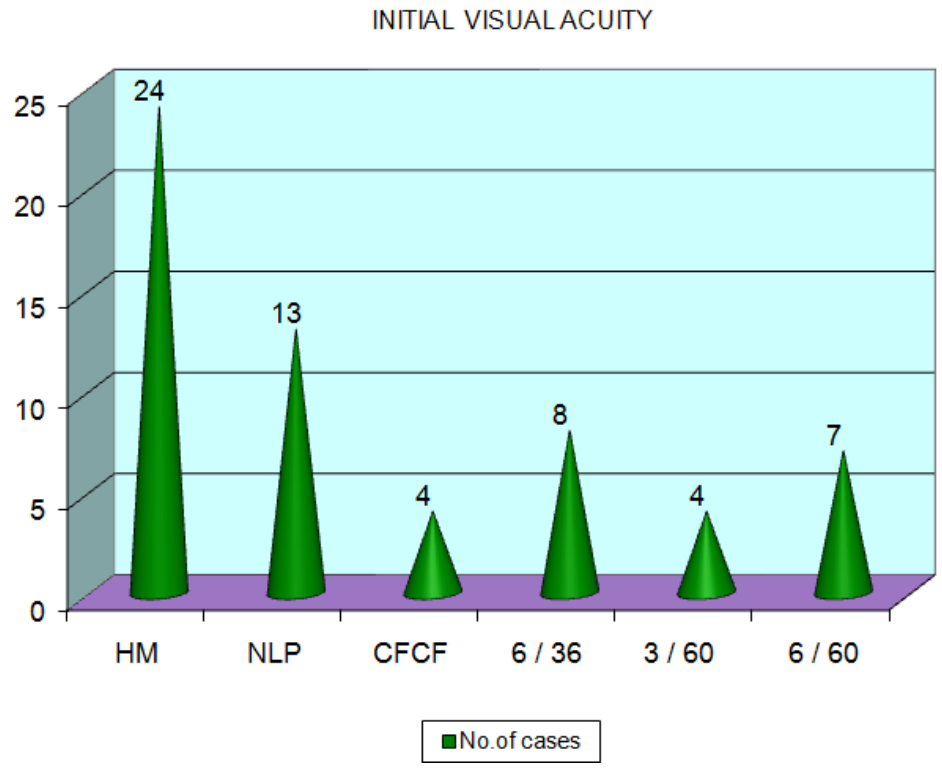
Time of presentation	No.of cases	Percentage
< 6	27	45.0
7 - 12	26	43.3
> 12	7	11.7
Total	60	100.0
Mean	7.4 hrs	



The mean duration between the admission and initiation of treatment after trauma was 7.4 hours.

TABLE 6: INITIAL VISUAL ACUITY

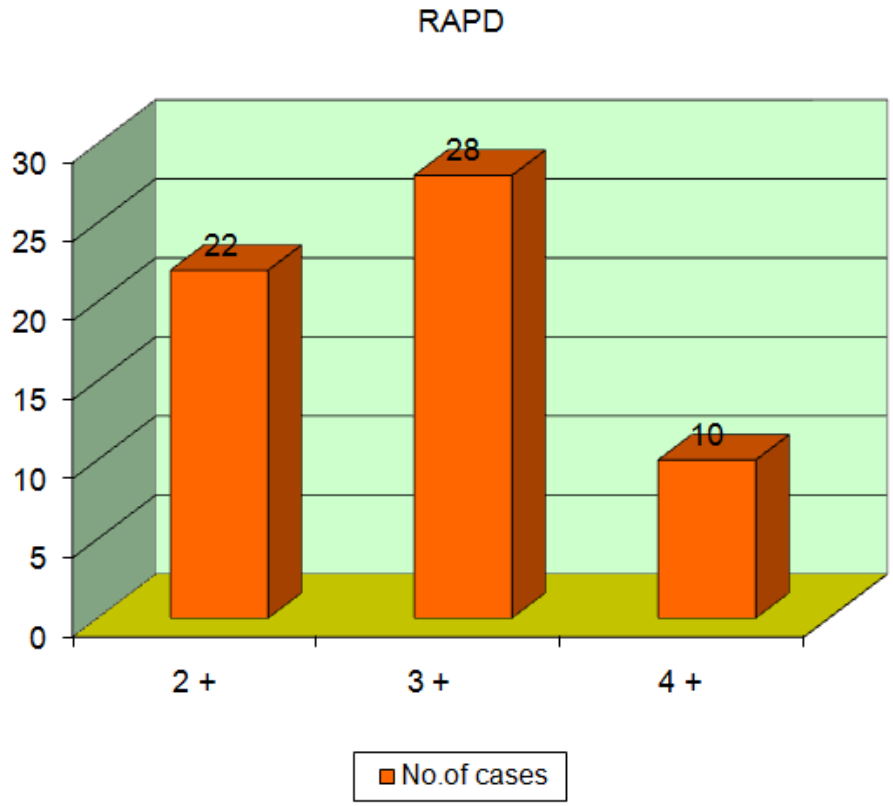
Initial visual acuity	No.of cases	Percentage
HM	24	40.0
NLP	13	21.7
CFCF	4	6.7
6 / 36	8	13.3
3 / 60	4	6.7
6 / 60	7	11.7
Total	60	100.0



Initial visual acuity ranged from NLP to 6/36

TABLE 7: RELATIVE AFFERENT PUPILLARY DEFECT

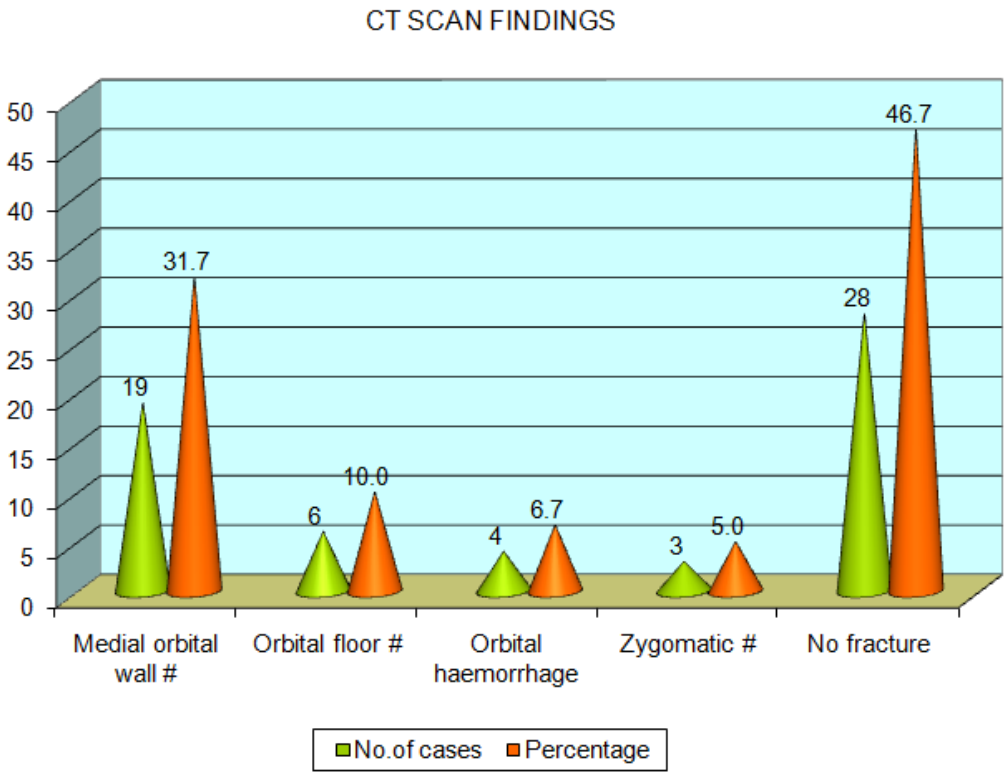
RAPD	No.of cases	Percentage
2 +	22	36.7
3 +	28	46.7
4 +	10	16.7
Total	60	100.0



100% patients in our study showed Relative afferent papillary defect (RAPD).

TABLE 8: CT SCAN FINDINGS

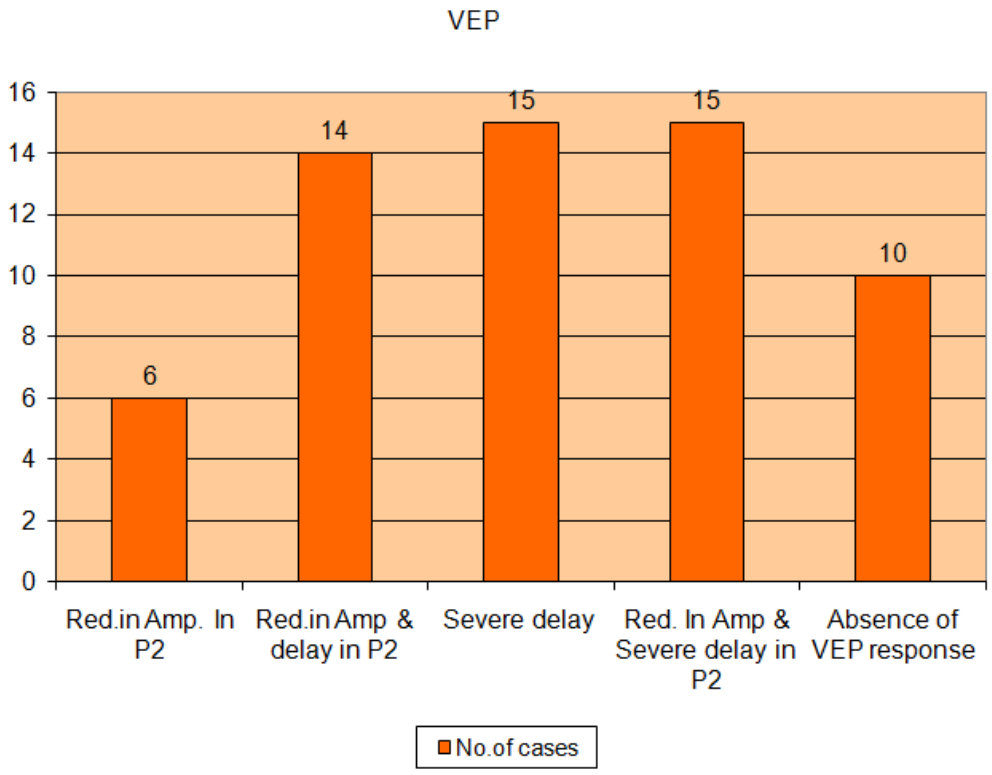
CT scan findings	No.of cases	Percentage
Medial orbital wall #	19	31.7
Orbital floor #	6	10.0
Orbital haemorrhage	4	6.7
Zygomatic #	3	5.0
No fracture	28	46.7
Total	60	100.0



Presence of orbital fracture on CT scan does not influence the visual outcome.

TABLE 9: VISUALLY EVOKED POTENTIAL

VEP	No.of cases	Percentage
Reduction in Amplitude In P2	6	10.0
Reduction in Amplitude and delay in P2	14	23.3
Severe delay	15	25.0
Reduction In Amplitude and Severe delay in P2	15	25.0
Absence of VEP response	10	16.7
Total	60	100.0



Severe delay in P2 conforms optic nerve injury. Absence of VEP response indicates poor visual prognosis.

TABLE 10: FINAL VISUAL OUTCOME

Final Visual Acuity	No.of cases	Percentage
NLP	11	18.3
HM	6	10.0
CFCF	11	18.3
3 / 60	4	6.7
6 / 60	14	23.3
6 / 12	8	13.3
6 / 9	6	10.0
Total	60	100.0

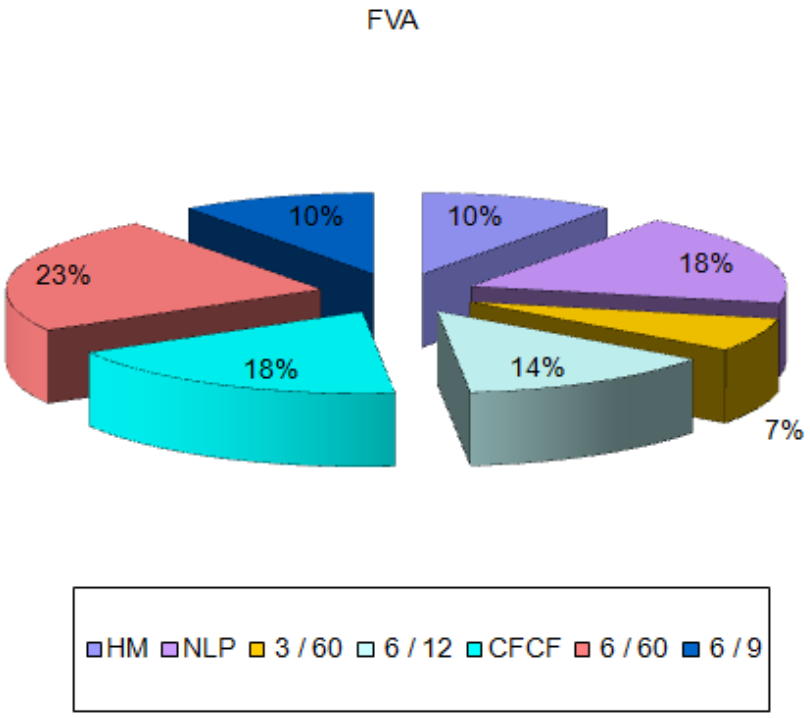


TABLE 11: COMPARISON OF INITIAL AND FINAL VISUAL ACUITY

Visual acuity	Initial visual acuity (No. of patients)	Final visual acuity (No. of patients)
NLP	13	11
HM	24	6
CFCF	4	8
3 / 60	4	9
6 / 60	7	14
6 / 36	8	6
6 / 9	0	6

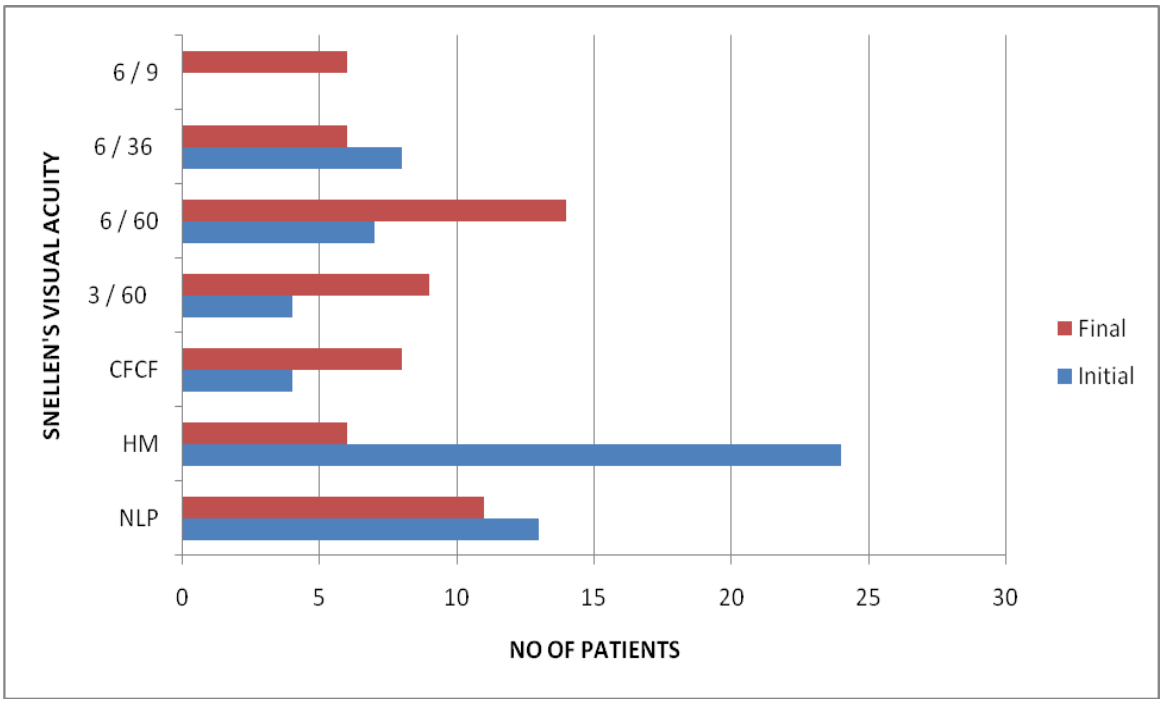


TABLE 12: STATISTICAL SIGNIFICANCE BY CHI SQUARE TEST

Pattern of VEP wave form	No. of cases	Final visual acuity 20 / <150
Reduction in P2 and Reduction Delay in P2	20	17
Reduction in amplitude and Severe delay, Severe delay and Absence of VEP	40	6
Chi square value	24.75	
p value	< 0.001 Significant	

Data were analysed using the statistical package for social sciences. The Chi square test was used for quantitative and qualitative data. P values <0.05 were considered statistically significant. pearson's coefficients were calculated for finding statistically significant relationships.

TABLE 13: DEGREE OF CORRELATION

	Correlation coefficient
Good correlation between RAPD vs initial visual acuity	-0.88
Good correlation between RAPD vs final visual acuity	-0.81
Poor correlation between orbital fractures and visual acuity	-0.38

SUMMARY

In order to establish the significant role of Visually Evoked Potential in early identification and prognosis of indirect optic neuropathy, 60 patients suspected to have traumatic optic nerve injury were subjected to Visually Evoked potential within 8 hours of trauma. Irrespective of the initial visual acuity ,all patients were invariably treated with IV Methyl Prednisolone 1 g OD for 3 days and subsequently followed with oral prednisolone 1mg/kg body weight/ day for 11 days and then tapered. All the patients' final visual acuity was assessed at the end of 2 weeks. 20 patients who showed reduced amplitude in P2 and both reduction in amplitude and delay in P2 had final visual acuity of >6/60 at the end of 2weeks, out of which 15 patients improved to >6/12. Out of the remaining 30 patients who showed severe delay only 6 patients improved to >6/60, indicating poor prognosis. 10 patients with absence of wave in VEP showed the worst prognosis.

To summarise:

- Baseline visual acuity is the most important predictor of final visual outcome.
- Visually evoked potential helped in early identification and confirmation of indirect traumatic optic nerve injury.
- Visually Evoked Potential also helped in predicting the prognosis and follow-up of patients with indirect traumatic optic nerve injuries.
- Visual recovery is impossible when Visually Evoked Potential waves are not recordable.

- Visually Evoked Potential guides in decision making regarding appropriate treatment.
- Relative afferent pathway defect is mainly related to the integrity of central vision, grading of RAPD, comparison of VEP amplitude between the affected and contralateral eyes useful for an objective evaluation of the visual functions of the affected eyes in patients with unilateral indirect optic nerve injuries.
- Presence of orbital fractures was not a predictive factor for initial or the final visual acuity.
- Patients who present within 8 hours of indirect traumatc optic nerve injuries with good initial isual acuity, Visually Evoked potential changes, lower grades of RAPD could predict better recovery of final visual acuity when treated with immediate high dose of steroid therapy.

DISCUSSION:

Indirect traumatic optic neuropathy is due to closed head trauma which results in transmission of force from forehead to the optic nerve in the optic canal or in the intracranial portion. It causes severe visual morbidity in the form of visual loss, RAPD, Colour vision defects, field defects and changes in visually evoked potential.

In our study clinical diagnosis made by pupillary examination was confirmed by visual evoked potential (VEP). VEP also quantifies the optic nerve dysfunction and predicts the prognosis earlier.

The average age of the patient is 30 years and ranges from 19-42 years. The gender ratio of male:female was 3:1. The higher male preponderance here was due to the fact that RTA, which is the commonest cause of optic nerve injury , occurs more in males.

Here almost 75% of patients had right eye involvement which was also statistically significant ($p<0.001$).

As expected Road traffic accidents (RTAs) were the commonest cause of Indirect traumatic optic nerve injury amongst which bike accidents were the most common. Fall from height being the next most common cause of this catastrophe.

The mean duration between admission and initiation of treatment after trauma was 7.4 hours (Range-7-8 hours).

The initial visual acuity ranged from NLP to 6/36. All patients in our study showed presence of RAPD.

Presence of orbital fracture in CT scan did not influence the final visual outcome.

Severe delay in P2 represents confirmation of optic nerve injury. Absence of VEP response indicates poor visual prognosis.

According to NASCIS III (National Acute Spinal Cord Injury Study) early treatment of steroids within 3-8hrs of injury and continuing 48 hrs were associated with better visual outcomes.

SOME PRACTICAL CHALLENGES IN OUR STUDY:

- There are often unavoidable delays in diagnosing TON when patients have life-threatening injuries that justifiably take precedence before an ophthalmological opinion is sought.

- If the patient is unconscious for a prolonged period, visual loss is likely to be reported late, and even if a clinical diagnosis is made within the 8-hour window, there are obvious ethical considerations to initiating potentially controversial treatment without proper informed consent.

- Each case therefore needs to be assessed on an individual basis, and the patient needs to be made fully aware of both the theoretical risks suggested by recent studies, and the real risks of a serious adverse event with active intervention.

CONCLUSION:

The study "**An observational study to evaluate the role of visually evoked potential in indirect traumatic optic nerve injuries and assessment of the visual outcome**" has given us the following conclusions.

Visual Evoked potential is a valuable clinical tool to confirm indirect optic nerve injury and to predict the visual prognosis following treatment. RAPD grades showed correlation with the results of Visual Evoked Potential.

PROFORMA

CASE NO. : MLC/Non-MLC DATE:

NAME : AGE : SEX:

OCCUPATION : IP No.: AR No.:

RIGHT EYE : LEFT EYE: BOTH EYE:

TIME OF INJURY:

MODE OF INJURY:

S.No.	COMPONENTS	YES	NO
1.	HISTORY OF LOC (IF YES DURATION)		
2.	HISTORY OF ENT BLEED		
3.	HISTORY OF CONVULSIONS		
4.	HISTORY OF TRANSIENT LOSS OF VISION		
5.	HISTORY OF PREVIOUS EYE PROBLEMS (IF YES SPECIFY)		
6.	HISTORY OF EYE SURGERY		
7.	HISTORY OF SMOKING		
8.	HISTORY OF ALCHOLISM		
9.	HISTORY OF DIABETES MELITUS		
10.	HISTORY OF SYSTEMIC HYPERTENSION		
11.	HISTORY OF UNDERLYING NEUROLOGICAL DISORDERS		

EXAMINATION

- INITIAL VISUAL ACUITY
- BEST CORRECTED VISUAL ACUITY
- VISUAL EVOKED POTENTIAL - LATENCY
- COLOUR VISION
- VISUAL FIELD DEFECT
- RAPD

OBLIQUE EXAMINATION

RIGHT EYE	STRUCTURE EXAMINED	LEFT EYE
	LIDS	
	CONJUCTIVA	
	CORNEA	
	ANTERIOR CHAMBER DEPTH	
	IRIS	
	PUPILS	
	LENS	
	EOM	
	ORBITAL MARGINS	

INTRAOCULAR PRESSURE AS MEASURED BY SHIOTZ TONOMETRY-

DILATED FUNDUS EXAMINATION:

OD		OS
	MEDIA	
	DISC	
	CUP-DISC RATIO	
	VESSELS	
	AV RATIO	
	MACULA	
	FR	

DIAGNOSIS:

FOLLOW UP

S.No.	COMPONENTS	AT THE TIME OF INJURY	3RD DAY	5TH DAY	7TH DAY	AFTER 2 WEEKS
1.	BCVA					
2.	RAPD					
3.	VEP					
4.	COLOR VISION					
5.	FIELD OF VISION					
6.	FUNDUS EXAMINATION					

ஆராய்ச்சி ஒப்புதல் படிவம்

தேதி:

ஆராய்ச்சி தலைப்பு:

பெயர்:

வயது:

உள்நோயாளி எண்:

பால்:

ஆராய்ச்சி சேர்க்கை எண்:

இந்த ஆராய்ச்சியின் விபரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை புரிந்துகொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

இந்தஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் நான் பங்குபெறுகிறேன், மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்தநேரமும் பின்வாங்கலாம் என்பதையும், அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும், நான்புரிந்துகொண்டேன்.

நான் என்னுடைய சுயநினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக் கொள்ள சம்மதிக்கிறேன் முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ என்னுடைய பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டார்கள் என்பதையும் அறிந்து கொண்டேன்.

எனது நோயின் தன்மை மற்றும் பின் விளைவுகளையும் முழுமையாக புரிந்து கொண்டேன். இந்த ஆராய்ச்சியில் எனது நோயின் மூலக்கூறு மற்றும் தன்மையை மட்டுமே ஆராய்வார்கள் என்பதை அறிந்துகொண்டேன்.

இதனால் என்வைத்திய முறைகளில் எந்தமாற்றமும் பார்வைத்திறனில் எந்தவித பாதிப்பும்ஏற்படாது என்பதையும் தெரிந்து கொண்டேன். எனக்கு விளக்கப்பட்ட விஷயங்களை முழுமையாக புரிந்து கொண்டு இந்த ஆராய்ச்சியில் பங்கு கொள்ள என் முழுமனதுடன் ஒப்புக்கொள்கிறேன்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்
தேதி:

S. No	Name	Age	Sex	Eye	Nature of Trauma	Time of presentation in hours	Initial VA	RAPD	CT Scan findings	VEP	Final Visual Acuity
1	Ramayee	33	F	R	Motor Accident	7	NLP	4+	Medial orbital wall#	Severe delay in P2	NLP
2	Kanthan	28	M	L	Accidental Fall	4	HM	3+	Zygomatic#	Reduction in amplitude and severe delay in P2	6 / 60
3	Shankar	27	M	L	Assault	6	CFCF	2+	No Fracture	Reduction in amplitude and delay in P2	CFCF
4	Vijaya	32	F	R	Fall from Height	4	NLP	4+	No Fracture	Reduction in amplitude and delay in P2	NLP
5	Stephen	23	M	R	RTA	8	HM	2+	No Fracture	Absence of VEP response	6 / 60
6	Nagamalaiyan	25	M	L	Fall from Height	3	HM	3+	Orbital haemorrhage	Reduction in amplitude and severe delay in P2	3 / 60
7	Madasamy	35	M	L	Trauma with stone	7	HM	3+	Medial orbital wall#	Reduction in amplitude and delay in P2	CFCF
8	Antonysamy	23	M	R	Car Accident	2	6/60	2+	Medial orbital wall#	Severe delay in P2	6 / 36
9	Murugan	32	M	L	Fall from Height	7	HM	3+	No Fracture	Severe delay in P2	CFCF
10	Jeeva	29	F	R	Bike Accident	5	6/36	2+	Medial orbital wall#	Severe delay in P2	6 / 12
11	Vijaya	40	F	L	Trauma with pipe	8	6 / 60	3+	Medial orbital wall #	Reduction in amplitude and severe delay in P2	6 / 12
12	Arasan	30	M	R	RTA	7	6 / 60	3+	No Fracture	Reduction in amplitude in P2	6 / 9
13	Pandiappan	27	M	R	RTA	12	NLP	4+	Orbital Floor#	Absence of VEP response	NLP

14	Karuthapandi	36	M	R	Assault	18	HM	3+	No Fracture	Reduction in amplitude and severe delay in P2	HM
15	Veerannan	24	M	R	RTA	20	CFCF	2+	No Fracture	Severe delay in P2	6 / 12
16	Lingeswaran	28	M	L	Accidental Fall	5	HM	3+	Zygomatic#	Reduction in amplitude and severe delay in P2	6 / 60
17	Raja	29	M	L	Assault	6	CFCF	2+	No Fracture	Reduction in amplitude and delay in P2	CFCF
18	Aarayee	32	F	R	Fall from Height	5	NLP	3+	No Fracture	Reduction in amplitude and delay in P2	NLP
19	Ravindran	26	M	R	RTA	9	HM	2+	No Fracture	Absence of VEP response	6 / 60
20	Muthusamy	24	M	L	Fall from Height	3	HM	3+	Orbital haemorrhage	Reduction in amplitude and severe delay in P2	3 / 60
21	Chinnasamy	35	M	R	Trauma with stone	6	HM	3+	Medial orbital wall #	Reduction in amplitude and severe delay in P2	CFCF
22	Kollu	25	F	R	Bike Accident	2	6 / 60	2+	Medial orbital wall #	Severe delay in P2	6 / 60
23	Pandian	32	M	R	Bike Accident	7	HM	3+	No Fracture	Severe delay in P2	CFCF
24	Murugesan	31	M	R	Bike Accident	5	6 / 36	2+	Medial orbital wall #	Severe delay in P2	6 / 12
25	Isakiammal	41	F	L	Trauma with pipe	8	6 / 60	2+	Medial orbital wall #	Reduction in amplitude and severe delay in P2	6 / 12
26	Pandi	30	M	R	RTA	6	6 / 60	3+	No Fracture	Reduction in amplitude in P2	6 / 9
27	Vadivel	34	M	R	RTA	12	NLP	4+	Orbital Floor#	Absence of VEP response	NLP

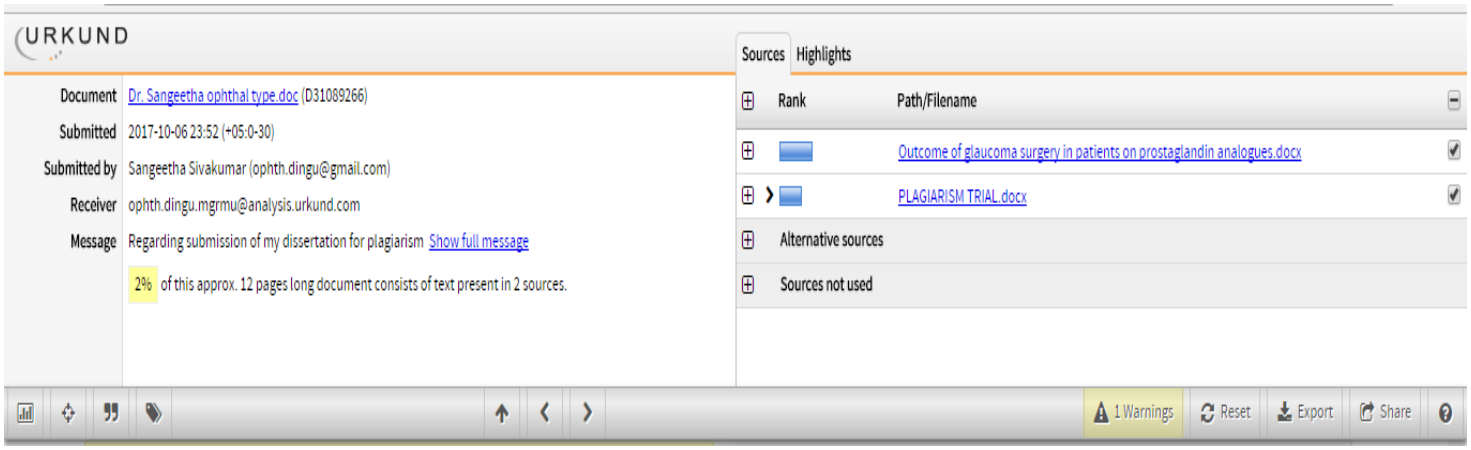
28	Sundaram	36	M	R	Assault	14	HM	3+	No Fracture	Reduction in amplitude and severe delay in P2	HM
29	Murugesan	31	M	R	RTA	7	CFCF	3+	No Fracture	Reduction in amplitude in P2	6 / 9
30	Fathima	28	F	R	RTA	13	NLP	3+	Orbital Floor#	Absence of VEP response	NLP
31	Suseela	27	F	R	Accidental Fall	18	HM	3+	No Fracture	Reduction in amplitude and severe delay in P2	HM
32	Chandran	25	M	R	RTA	15	CFCF	2+	No Fracture	Severe delay in P2	6 / 12
33	Baskaran	33	M	R	Bike Accident	4	NLP	3+	No Fracture	Reduction in amplitude in P2	NLP
34	Reeta	33	F	R	Motor Accident	9	NLP	4+	Medial orbital wall #	Severe delay in P2	NLP
35	Mahalingam	28	M	L	Accidental Fall	4	HM	3+	Orbital haemorrhage	Reduction in amplitude and severe delay in P2	6 / 60
36	Raman	31	M	L	Assault	6	CFCF	2+	No Fracture	Reduction in amplitude and delay in P2	CFCF
37	Iyemmal	32	F	R	Fall from Height	5	NLP	4+	No Fracture	Reduction in amplitude and delay in P2	NLP
38	Nandagopalan	23	M	R	RTA	8	HM	3+	No Fracture	Absence of VEP response	6 / 60
39	Srinivasan	25	M	L	Fall from Height	4	HM	2+	Orbital haemorrhage	Reduction in amplitude and severe delay in P2	3 / 60
40	Ramasamy	34	M	R	Trauma with stone	8	HM	2+	Medial orbital wall #	Reduction in amplitude and delay in P2	CFCF

41	Chinthamani	23	F	R	Car Accident	3	6 / 60	2 +	Medial orbital wall #	Severe delay in P2	6 / 60
42	Baskaran	31	M	L	Fall from Height	7	HM	3 +	No Fracture	Severe delay in P2	CFCF
43	Chandran	32	M	R	Bike Accident	5	CFCF	2 +	Medial orbital wall #	Reduction in amplitude and delay in P2	CFCF
44	Karuppan	40	M	R	Trauam with pipe	7	6 / 60	3 +	Medial orbital wall #	Reduction in amplitude and severe delay in P2	6 / 12
45	Ramasamy	32	M	R	RTA	7	6 / 60	3 +	No Fracture	Reduction in amplitude and delay in P2	6 / 9
46	Andichi	27	F	R	RTA	11	NLP	4 +	Orbital Floor #	Absence of VEP response	NLP
47	Alagambalam	36	M	R	Assault	16	HM	3 +	No Fracture	Reduction in amplitude and severe delay in P2	HM
48	Kaliammal	28	F	R	Bike Accident	7	NLP	4 +	Medial orbital wall#	Severe delay in P2	NLP
49	Karuppan	28	M	R	Accidental Fall	4	HM	3 +	Zygomatic#	Reduction in amplitude and severe delay in P2	6 / 60
50	Ramasamy	27	M	R	Assault	5	CFCF	2 +	No Fracture	Reduction in amplitude and delay in P2	CFCF
51	Avammal	32	F	R	Fall from Height	4	NLP	4 +	No Fracture	Reduction in amplitude and delay in P2	NLP
52	Sivakumar	26	M	R	RTA	8	HM	2 +	No Fracture	Absence of VEP response	6 / 60
53	Krishnan	24	M	R	Fall from Height	5	HM	2 +	Orbital Floor#	Absence of VEP response	NLP

54	Petichamma l	35	F	L	Trauma with stone	7	HM	3+	Medial orbital wall #	Reduction in amplitude and delay in P2	CFCF
55	Thamarai	23	M	R	Car Accident	2	3 / 60	2+	Medial orbital wall #	Severe delay in P2	6 / 60
56	Isakimuthu	33	M	L	Car Accident	7	HM	3+	No Fracture	Reducation in amplitude in P2	6 / 9
57	Kannan	29	M	R	Bike Accident	5	3 / 60	2+	Medial orbital wall #	Severe delay in P2	6 / 12
58	Aarayee	42	F	R	Trauma with pipe	9	CFCF	3+	Medial orbital wall#	Reduction in amplitude and severe delay in P2	6 / 12
59	Brindha	30	F	R	RTA	8	6 / 60	2+	No Fracture	Reducation in amplitude in P2	6 / 9
60	Kajamohideen	29	M	R	RTA	11	NLP	4+	Orbital Floor #	Absence of VEP response	NLP

KEY TO MASTER CHART

R-RIGHT
L-LEFT
M-MALE
F-FEMALE
RTA-ROAD TRAFFIC ACCIDENTS
NLP-NO LIGHT PERCEPTION
HM-HAND MOVEMENTS
CFCF-COUNTING FINGERS CLOSE TO FACE
VA-VISUAL ACUITY
RAPD-RELATIVE AFFERENT PUPILLARY DEFECT
CT-COMPUTERISED TOMOGRAPHY
#-FRACTURE
VEP-VISUAL EVOKED POTENTIAL
IP-INPATIENT
AR- ACCIDENT REGISTER
MLC-MEDICO LEGAL CASE
EOM-EXTRA OCULAR MOVEMENTS
A:V-ARTERY:VEIN
FR FOVEAL REFLEX


URKUND
Document Dr. Sangeetha ophthal type.doc (D31089266)
Submitted 2017-10-06 23:52 (+05:0-30)
Submitted by Sangeetha Sivakumar (ophth.dingu@gmail.com)
Receiver ophth.dingu.mgrmu@analysis.urkund.com
Message Regarding submission of my dissertation for plagiarism Show full message
2% of this approx. 12 pages long document consists of text present in 2 sources.
Sources Highlights
Rank
Path/Filename
Outcome of glaucoma surgery in patients on prostaglandin analogues.docx
PLAGIARISM TRIAL.docx
Alternative sources
Sources not used
1 Warnings
Reset
Export
Share

MADURAI MEDICAL COLLEGE
MADURAI, TAMILNADU, INDIA -625 020
(Affiliated to The Tamilnadu Dr.MGR Medical University, Chennai, Tamil Nadu)

ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.D.Sangeetha
Course	:	PG in MS., Ophthalmology
Period of Study	:	2014 - 2017
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	An observational study to evaluate the role of visual evoked potential in indirect traumatic optic nerve injuries and assessment of visual outcome
Ethical Committee as on	:	27.07.2017

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary

Prof Dr V Nagaraajan
M.D., MNAMS, D.M.(Neuro), Dsc (Hon)
CHAIRMAN
IEC - Madurai Medical College
Madurai

Dean / Convenor

DEAN
Madurai Medical College
Madurai-20